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SOCIETY FOR VASCULAR SURGERY ISSUE

Vol. 41 JANUARY, 1957 No. 1

SURGERY

A Monthly Journal Devoted to the Art and Science of Surgery

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SURGERY

VOL. 41 JANUARY, 1957 No. 1

Original Communications

Society for Vascular Surgery

THE FIRST TEN YEARS OF THE SOCIETY FOR VASCULAR SURGERY

GEORGE D. LILLY, M.D., MIAMI, FLA.

(From the Surgical Service of the Victoria Hospital)

ON DEC. 5, 1945, a group of general surgeons who were interested in the field of vascular surgery held an informal conference during the meeting of the Southern Surgical Association at Hot Springs, Virginia. Those in attendance were: Arthur Allen, Isaac Bigger, Arthur Blakemore, Barney Brooks, Mims Gage, George Lilly, Alton Ochsner, and Ross Veal. The meeting was arranged by Dr. Veal, who, for several years, had been advocating the formation of a new society composed of those physicians interested in the development of surgery of the cardiovascular system. Many of the problems involved were discussed, and plans were made to hold an organizational caucus during the meeting of the American Medical Association in San Francisco in July, 1946. Two recollections of this first meeting stand out in my memory: Dr. Brooks pointed out that surgery of the peripheral vascular system constituted only a part of the field, and that surgery of the heart and great vessels would make rapid strides within the near future. Dr. Arthur Allen commented that one of his associates at the Massachusetts General Hospital, Dr. Robert Linton, was on the verge of perfecting a procedure to relieve postthrombophlebitic edema.

On July 3, 1946, an informal meeting was held at the Fairmont Hotel in San Francisco. In attendance were: Michael De Bakey, Geza de Takats, Daniel Elkin, Norman Freeman, Emile Holman, John Homans, Robert Linton, Alton Ochsner, and Ross Veal. These men selected as the name for the new organization "The Society for Vascular Surgery," and the following officers were elected: president, Dr. Alton Ochsner; vice-president, Emile Holman; recorder, Geza de Takats; secretary, J. Ross Veal.

Presidential address, read at the tenth annual meeting of the Society for Vascular Surgery, Chicago, Ill., June 10, 1956.

Thirty-one charter members were selected: Drs. Arthur Allen, Frederick Bancroft, Claude Beck, Isaac Bigger, Arthur Blakemore, Alfred Blalock, Barney Brooks, Frederick Coller, Michael De Bakey, Geza de Takats, Daniel Elkin, Norman Freeman, Mims Gage, Robert Gross, Louis Herrmann, Emile Holman, John Homans, Lucian Landry, George Lilly, Robert Linton, Rudolph Matas, Walter Maddock, John Morton, Alton Ochsner, Herman Pearse, Rawley Penick, Merle Scott, Harris Shumacker, Reginald Smithwick, Ambrose Storck, and Ross Veal.

The first annual meeting was scheduled for June, 1947, at Atlantic City, and Drs. Allen, Linton, Holman, Blalock, and Gage were appointed as a program committee to cooperate with Dr. Veal in the preparation of a suitable program.

On the morning of June 8, 1947, the first annual meeting of The Society for Vascular Surgery was called to order by Dr. Alton Ochsner. This meeting was held at the Hotel Dennis in Atlantic City. The morning was devoted to organizational problems and plans for the future. It was decided that the annual meeting would be held on the Sunday preceding the meeting of the American Medical Association, and in the same city; this was done to enable members to combine attendance of the two meetings into a single trip, and because it was desired that the meeting would be open to nonmember physicians who might wish to come to the American Medical Association a day early and attend this meeting. A committee was appointed to draw up a constitution and by-laws for the Society. Dr. Walter Maddock was the chairman, and the other members of this committee were: Drs. Coller, De Bakey, and Holman.

New officers elected to serve for the 1947-48 term were: Dr. Arthur Allen, president; Dr. Isaac Bigger, vice-president; Dr. Geza de Takats, recorder; Dr. Harris Shumacker, treasurer, and Dr. J. Ross Veal, secretary.

The afternoon of the first meeting was devoted to a scientific session, at which nine papers were presented:

Alton Ochsner:	(Presidential Address) "Venous Thrombosis"
Robert R. Linton:	"Postthrombophlebitic States of the Lower Extremity. Treatment by Superficial Femoral Vein Interruption and Ligation and Stripping of Long and Short Saphenous Veins."
Arthur Blakemore:	"Portal Hypertension. Indications for Portocaval Anastomosis"
Alfred Blalock:	"Congenital Pulmonic Stenosis"
Robert E. Gross:	"Surgical Treatment of Coarctation of the Aorta"
Frank Gerbode:	"Experimental Studies in Cardiac Hypertrophy"
Harris Shumacker, Jr.:	"Causalgia"
Michael De Bakey:	"Hemometakinesia and Its Therapeutic Significance in Peripheral Vascular Disease"
Geza de Takats:	"The Surgical Treatment of Essential Hypertension"

It is interesting to note that only three of the papers listed coauthors that year. This is in strange contrast to the usual "departmental paper" of today, whose list of authors looks more like a board of directors.

The evening program of the Society's first meeting consisted of a grand panel discussion, presided over by Dr. Arthur Allen. Written questions were directed to the various members of the Society by the medical audience, and a general discussion was conducted.

On June 20, 1948, the second annual meeting was held at the Stevens Hotel in Chicago. Dr. Arthur Allen presided and opened the morning program with "An Evaluation of the Prophylaxis and Treatment of Venous Thrombosis and Pulmonary Embolism." The second place on the program was occupied by Dr. John Homans, who, at that time, presented his first and last paper before this Society. The title of his paper was "The Management of Recovery from Venous Thrombosis in the Lower Limbs."

A total of fifteen original papers were presented, and twenty-three of the thirty-one members were in attendance. Five hundred nonmember physicians attended the meeting.

At the second meeting, Dr. Veal introduced a gavel which he had designed from a piece of oak from the entrance railing of the old Charity Hospital of New Orleans, where Dr. Rudolph Matas had done so much of his early work in the field of vascular surgery. The president presented this gavel to Dr. Matas who accepted it and immediately gave it to the Society. It was named "The Matas Gavel" and was designated as the official gavel of the Society. The following officers were elected: Dr. Emile Holman, president; Dr. Reginald Smithwick, vice-president; Dr. Geza de Takats, recorder; Dr. Harris Shumacker, treasurer; and Dr. J. Ross Veal, secretary.

The third annual meeting was held on June 5, 1949, in the Dennis Hotel, Atlantic City. Dr. Holman presided, and twelve papers were presented. Of particular interest was a paper by Drs. Julian Johnson and Charles Kirby entitled "The Experimental Clinical Use of Vein Grafts to Replace Large Arteries." This is the first paper to be presented before this Society on the subject of vessel grafts.

At this meeting Dr. Walter Maddock presented his committee's proposed constitution and by-laws, and these were adopted by the membership. Article II of the Constitution might well be emphasized at this point; it read as follows:

> The objects of this Society shall be (1) to promote the study of and research in vascular diseases; (2) to define more clearly the role of surgery in these diseases; (3) to pool the experience and knowledge of the membership, in order to standardize methods of studying and managing these diseases; (4) to standardize the nomenclature of these diseases; (5) to promote and encourage adequate teaching of these diseases to students, internes and residents. (6) to encourage hospitals to develop special training for young surgeons interested in this field; and, (7) to hold annual meetings.

Officers elected for the ensuing year were: Dr. Daniel Elkin, president; Dr. Louis Herrmann, vice-president; Dr. Geza de Takats, recorder; Dr. Harris Shumacker, treasurer; and Dr. J. Ross Veal, secretary.

A special category of foreign corresponding membership was created, and the following were elected to this type of membership: Professor Rene Leriche, Paris; Professor Rognvald Learmonth, Edinburgh; Dr. Clarence Crafoord, Stockholm; Professor Rene Fontaine, Strasbourg; and Dr. Ferdinand Martorell, Barcelona.

On June 25, 1950, the fourth annual meeting was held at the Fairmont Hotel in Chicago, where the Society had been organized in 1946. Dr. Daniel Elkin presided, and thirteen papers were presented. Four honorary members were elected; these were: Dr. Rudolph Matas, Dr. John Homans, Dr. Paul Moure, and Dr. Rene Leriche. At this meeting Dr. De Bakey proposed an amendment to the by-laws, creating an advisory board consisting of the four immediate living past presidents of the Society. This advisory board was authorized to act in an advisory capacity in connection with any changes in policy which may come before the Council or the membership of the Society.

Dr. Emile Holman offered an amendment to the by-laws, creating the John Homans Lecture. This specified that, from time to time, when deemed advisable by the Council, eminent investigators in the field of vascular surgery or allied sciences may be invited to present the John Homans Lecture during the annual meeting.

Dr. Daniel Elkin presented the first Homans Lecture the following year (1951). The title of his presentation was "John Homans: The Man and His Work." This was published in the *New England Journal of Medicine* in December of that same year. This afternoon, we shall hear the second Homans Lecture, which will be presented by Professor Rene Fontaine, of Strasbourg.

At the 1950 meeting, Dr. Veal, the man who conceived the idea of creating the society and who served as its all-important secretary during the first four years of its existence, was elected president. Other officers elected were: Mims Gage, vice-president; George Lilly, secretary; Geza de Takats, recorder; and Harris Shumacker, treasurer.

The fifth annual meeting took place at Atlantic City on June 10, 1951. Dr. Veal presided. Fifteen scientific papers were presented by members of the society. The title of Dr. Veal's presidential address was "Diseases of the Superior Vena Caval System." Officers elected that year were: Alfred Blalock, president; Arthur Blakemore, vice-president; George Lilly, secretary; Harris Shumacker, treasurer; and Geza de Takats, recorder.

The sixth annual meeting of the Society was held at the Conrad Hilton Hotel in Chicago, on June 8, 1952. Dr. Blalock presided, and the subject of his presidential address was "The Anatomy of Pulmonary Stenosis and Atresia, with Comments on Surgical Therapy." A total of sixteen papers were presented; five of these dealt with blood vessel grafts. At this meeting, Dr. Shumacker presented the first paper dealing with intracardiac surgery. His title was "Failure of Transventricular Autogenous Tubes as Suitable Means for Diminishing Valvular Insufficiency."

Officers elected at this meeting were: Geza de Takats, president; Robert Gross, vice-president; George Lilly, secretary; Harris Shumacker, treasurer; and Frank Gerbode, recorder.

On May 31, 1953, the seventh meeting was held at the Hotel Pierre in New York, with Dr. de Takats presiding. The title of his presidential address was "Anticoagulant Therapy." It is most interesting to note that at this meeting the only paper on varicose veins to be presented before this Society was given by Dr. Luke. In spite of the deaccentuation of this lowly subject, I am quite sure that it is the field in which members of this Society have afforded mankind the greatest relief from suffering. At this meeting, Dr. De Bakey and Dr. Cooley presented their early work on excisions of aneurysms of the thoracic aorta and great vessels. Officers elected for the 1953-54 period were: Michael De Bakey, president; Louis Herrmann, vice-president; George Lilly, secretary; George Yeager, treasurer; and Frank Gerbode, recorder.

On June 30, 1954, the eighth annual meeting was held at the Mark Hopkins Hotel in San Francisco, with Dr. De Bakey presiding. Twenty scientific papers were presented, and the total attendance exceeded 800 physicians. Officers elected were: Robert Linton, president; Keith Grimson, vice-president; George Lilly, secretary; George Yeager, treasurer, and Frank Gerbode, recorder. The wonderful entertainment which Dr. Gerbode provided for the members of the Society will be remembered for many years.

The ninth meeting was held in the same room as the first and third meetings, at the Hotel Dennis in Atlantic City, on June 5, 1955. Dr. Linton presided, and the subject of his address was "Some Practical Considerations of Blood Vessel Surgery." Seventeen papers were presented. At this meeting the Council created a committee for the study of vascular prosthesis, whose first report you have heard this morning. The officers elected at this meeting were: George Lilly, president; Julian Johnson, vice-president; Henry Swan, secretary; George Yeager, treasurer, and Frank Gerbode, recorder.

During this short period of ten years, the members of this Society have had the privilege of participating in a tremendous expansion of the field of cardiovascular surgery. Techniques for the correction of intracardiac lesion and for the replacement of diseased great vessels have been perfected, and have been established as practical for clinical application. The future possibilities in this field are, indeed, unlimited. And I am quite sure that with the perfection of apparatus for the carrying on of extracorporal circulation, which was so patiently developed by Dr. Gibbon, even greater feats will be accomplished within the near future.

As a whole, I feel that the modern vascular surgeon is to be complimented on his cautious and conservative approach to new procedures and on his untiring experimental work in the animal laboratories before applying new techniques to the correction of human ailments. Undoubtedly, however, all of us will be tempted from time to time to short-circuit tedious animal experimentation and apply new theories to clinical practice. It is for this reason that I should like to remind our members of the famous admonition which Dr. Harvey Stone made while discussing the report of a radical cancer surgeon: "The modern surgeon must ever bear with him a scrupulous and conscientious caution, lest he become a modern Charon, exacting an exorbitant toll for that final trip across the River Styx."

THE JOHN HOMANS' MEMORIAL LECTURE

REMARKS CONCERNING VENOUS THROMBOSIS AND ITS SEQUELAE

RENÉ FONTAINE, M.D., STRASBOURG, FRANCE

(From the Université de Strasbourg, Clinique Chirurgicale "A," and the Raymond-Poincaré Laboratory for Surgical Research)

INTRODUCTION

DR. LOUIS G. HERRMANN (Cincinnati, Ohio).—The problem of the treatment of venous thrombosis and its sequelae was John Homans' major interest during the latter decades of his life. In spite of the many valuable contributions to this problem from all parts of the world, there still remains much to be learned about this many-faceted problem.

The special committee, which our President appointed to select an outstanding vascular surgeon from across the Atlantic to present the second John Homans' Memorial Lecture, has been hard at work for several months arranging for this part of our program. It was the unanimous opinion of the members of this committee that the John Homans' Lecture should deal with some recent advances in our knowledge of venous thrombosis and its complications.

We are highly honored this afternoon to have as our lecturer a man who has made many valuable contributions to many fields of vascular surgery. He is the outstanding pupil and former associate of the late René Leriche, an Honorary Member of our Society. He is known throughout Europe and the Near East for his work as a surgeon, as a teacher, and as an investigator. He was Chief Surgeon of the Resistance Forces in France after her surrender in 1940 and he has continued to be an adviser to the Surgeon General of the French Army in the capacity of Chief Consulting Surgeon of the Eastern Division.

We are privileged today to welcome to this meeting the distinguished Dean of the Faculty of Medicine and Professor of Surgery of the University of Strasbourg, France. It gives me real pleasure to present to you my old friend and colleague, who will now present the second John Homans' Memorial Lecture. His subject is the Problem of Venous Thrombosis and Its Complications.

I wish to present to you Professor René Fontaine of France.

IN THIS present era when progress in the medical sciences appears to be so rapid, our young surgeons frequently overlook the fundamental contributions which have been made by surgeons of past generations. They often consider the work of earlier surgeons as a form of dilettantism and a product of the "good old days," which the Germans love to call "Bidermaierzeit." They also have the false impression that in those days the problems were simple and the solutions easy; consequently, there was plenty of time for meditation and introspection and even leisure to cultivate an added interest in the arts and in the humanities.

Presented at the tenth annual meeting of the Society for Vascular Surgery, Chicago, Ill., June 10, 1956.

Young surgeons of today often express the belief that the only difference between scientific research of yesteryear and that of today is the degree of profoundness of the studies. Surgeons of the past are thought to have had a broad general education of the encyclopedic type, but with a distinct tendency to be superficial; while today the surgeon prides himself on his thorough knowledge in only a small field of science with little or no understanding of surgical diseases in general. It is easy, therefore, to understand how many specialists of today, engrossed in the newer techniques which have evolved from previous work, have a tendency to forget the monumental work of men of the past and, therefore, fail to recognize the real debt we all owe these pioneers of research and practice in surgery.

How many of us think of the pioneers who struggled hard to clear the forests and build primitive roads, as we now cruise in our automobiles at high speed over these same hills on modern superhighways? When we fly from one continent to another in a few hours, we rarely take time to reflect about the men who suffered or lost their lives to make such transportation possible.

A few weeks ago as I sat in my study writing this address, I gazed out the window at the great cathedral of Strasbourg. I was enthralled by the purity of its architecture, the gracefulness of its high arches, the richness of its decorations, and the perfection of its world-famous clock. Suddenly I realized that no one living today remembers the humble workmen who built its foundations or buttressed the great walls which have for centuries withstood the ravages of the elements and the cataclysms caused by the stupidity and wickedness of men.

Thus men of one generation too often think only of the accomplishments of their own time. Because of this great capacity of man to forget the earlier struggles and advances which make our present-day accomplishments possible, I believe we should regularly take time to pay homage to our illustrious predecessors and express our gratitude for the foundation stones which they have provided for us.

I am intrigued by the American custom of placing this scientific gathering under the sponsorship of one of our great predecessors in the field of vascular surgery. By dedicating our studies to such a leader of the past generation, we provide this opportunity to reiterate the lasting value of his teachings. His spiritual presence at this meeting should act as a powerful stimulus and a guide to keep our search for scientific truths far away from excessive optimism and unnecessarily fruitless pessimism, the two greatest deterents to the success of this search.

We are here today to honor the memory and work of John Homans. You have graciously entrusted this important task to me, and I have accepted the invitation in the name of all French surgeons who knew and admired John Homans. I assure you it is a great honor for me to speak to you on behalf of my colleagues.

It was Professor René Leriche, my chief, who first aroused my interest in the work of John Homans, and it was Elliott Cutler who made our first meeting in 1928 possible. I was an exchange assistant resident on the Surgical

Service of Dr. Cutler in Cleveland in that year, and I welcomed the opportunity of visiting John Homans at work in Boston. Up to that time I had known him only through his scientific writings. My friends described him as a friendly, distinguished, and learned surgeon, an experienced clinician, and a skilled investigator. At times his words were caustic, but he always showed patience and understanding when he talked with younger surgeons. I had been told that he had a deep love for his work, for he came from a family of distinguished physicians. Surgeons in every part of the world praised the perfection of his technical methods, and everyone admired the clarity of his teachings. Such was the picture I had been given of John Homans. When I saw him, this description of the man was only surpassed by the pleasure of meeting him. I was greatly impressed by this little man with his frank, energetic, and forthright look. His gestures were precise but restrained; his speech was deliberate, at times cutting but never offending; and his words were tempered by a profound human interest born of his long family traditions. The work of John Homans is an excellent reflection of the man, John Homans. His many contributions to scientific literature showed the wide variety of his interests. His vast store of knowledge predestined him to write his *Textbook of Surgery,* which was an immediate success and became the bedside guide for hundreds of young surgeons in my country.

The pathologic changes in peripheral veins seemed to be his prime interest, and his keen and piercing clinical sense made him realize the importance of pain in the calf muscles on flexion of the foot in patients with deep venous thrombosis. This sign bears his name today. He was always a careful observer and his monograph on *Circulatory Diseases of the Extremities,* which he first published in 1939, placed him among the undisputed leaders in the field of vascular surgery.

In addition, we honor the memory of a man whose modesty was as great as his ability, and the brilliant example which he set forth should shine over the meetings of this Society for many years to come.

I wish therefore to dedicate this humble work to the memory of my friend, John Homans.

VENOUS THROMBOSIS

There are so many facets to this great problem that I hope you will understand if I discuss only those aspects which we have studied extensively in our laboratories and clinics during the past two decades.

Phlebothrombosis and Thrombophlebitis: Single or Multiple Etiology.— Since the work of Virchow, which initiated the modern investigation of the process of venous thrombosis, we have all come to consider the following three factors as being of etiologic significance:

1. Structural changes in the wall of the vein
2. Venous stasis
3. Changes in the constituents of the blood

In recent years the greatest emphasis has been placed on the changes in the blood as the etiologic factor of venous thrombosis with the result that the other two factors have been somewhat neglected.

Endothelial changes in veins were first described by Bizzozero in 1882 and are considered by almost all investigators as being important; yet they are extremely difficult to demonstrate. Géry has described these macroscopic changes as slight scratches on the intima in the emptied vein which was the source of a clot that caused fatal pulmonary embolism. They are rarely visible under the microscope in the routine sections made from the vein; yet we have, by special techniques, demonstrated that these changes do occur frequently. We have also interpreted some of the minor changes in the intima as a response to stress, and we have shown them to exist in peripheral veins after surgical operations, after childbirth, and in patients with acute and chronic diseases.

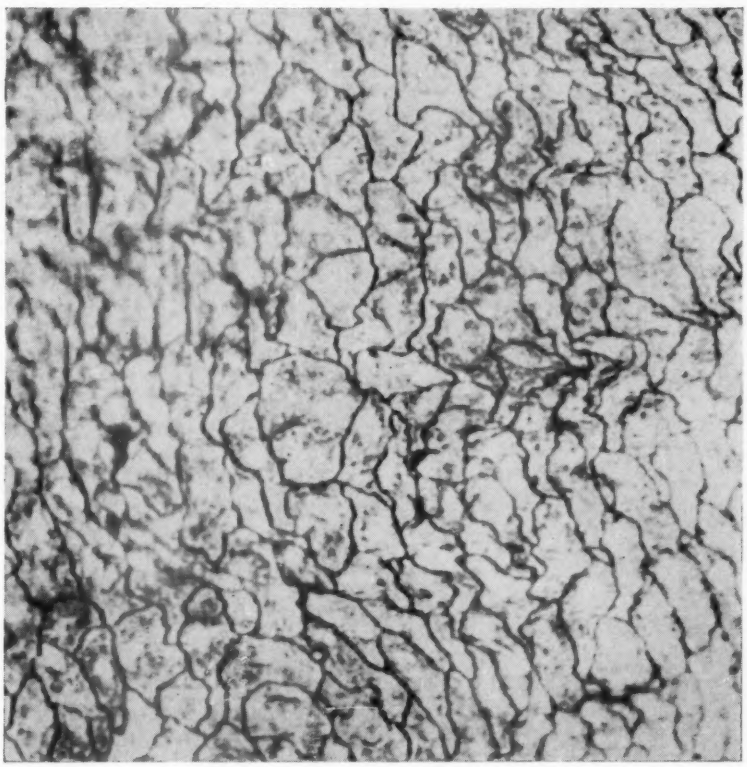

Fig. 1.—A normal vein showing the endothelium as demonstrated by the silver nitrate method.

Fonio, Roskam, and others have shown that the roughing of the endothelial lining of veins is common, and almost always a small platelet clot forms at the site of the injury, but these changes do not necessarily lead to clinical venous thrombosis. They point out that these changes are evidence of extreme fragility of the endothelium of veins.

Two members of my staff, C. Bollack and B. Winisdoerffer, carried this problem to the laboratory, and they found in their studies of stretched portions of the endothelium of veins of dogs prepared by the silver nitrate method that these endothelial injuries were present. Even the injection of 10 per cent sodium chloride solution into the segment of the vein isolated by the ligatures will likewise cause the endothelial cells to swell very quickly, become vacuolated, and finally desquamate in patches of various sizes. They showed that

trivial injury, both chemical and mechanical, could cause extensive changes in the intima of veins and thus set the stage for the development of venous thrombosis.

It is our impression, however, that these endothelial changes alone are not sufficient to account for all instances of venous thrombosis, and we believe we have evidence to show that there exists a kind of balance between the endothelial changes, the degree of stasis of blood, and the composition of the blood.

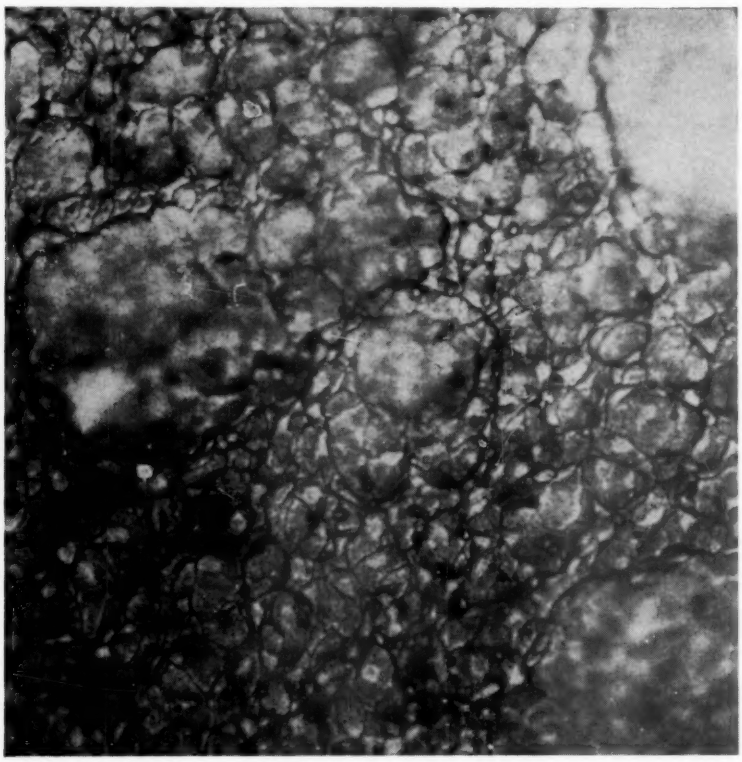

Fig. 2.—Phlyctenular change in the endothelial cells following denudation of the vein (silver nitrate method).

We have come to the conclusion, therefore, that one of these factors alone would not precipitate serious venous thrombosis, and that is one reason why we do not believe the phlebothrombosis of Ochsner and De Bakey and the thrombophlebitis of the ordinary clinical variety need be considered as different pathologic entities.

In clinical practice, however, it is undeniably true that there exist two degrees of venous thrombosis: one which Homans called "bland thrombosis," characterized by minimal local signs and symptoms and with great danger of producing pulmonary embolism; and the other which produces the typical form of phlegmasia alba dolens with little danger of pulmonary embolism. I believe that the obstructing clot within a vein is formed in the same way in all instances. Starting with a small accumulation of platelets and cells at the site of injury to the intima, this clot continues to develop and produce a white clot, which later is changed to the characteristic red clot of venous stasis. For some years, I have been of the opinion that the clot within a vein completely

fills the vein immediately when the venous thrombosis takes place; but after this first phase of coagulation which lasts only a very short time, the retraction of the clot begins to take place. If this retraction of the clot is pronounced, the clot will be separated from the walls of the vein and a firm nonobstructing clot will be produced. This clot is always attached at one point somewhere in the vein and it is this free-floating portion of the clot which can be dislodged easily and transported to the pulmonary circulation in the form

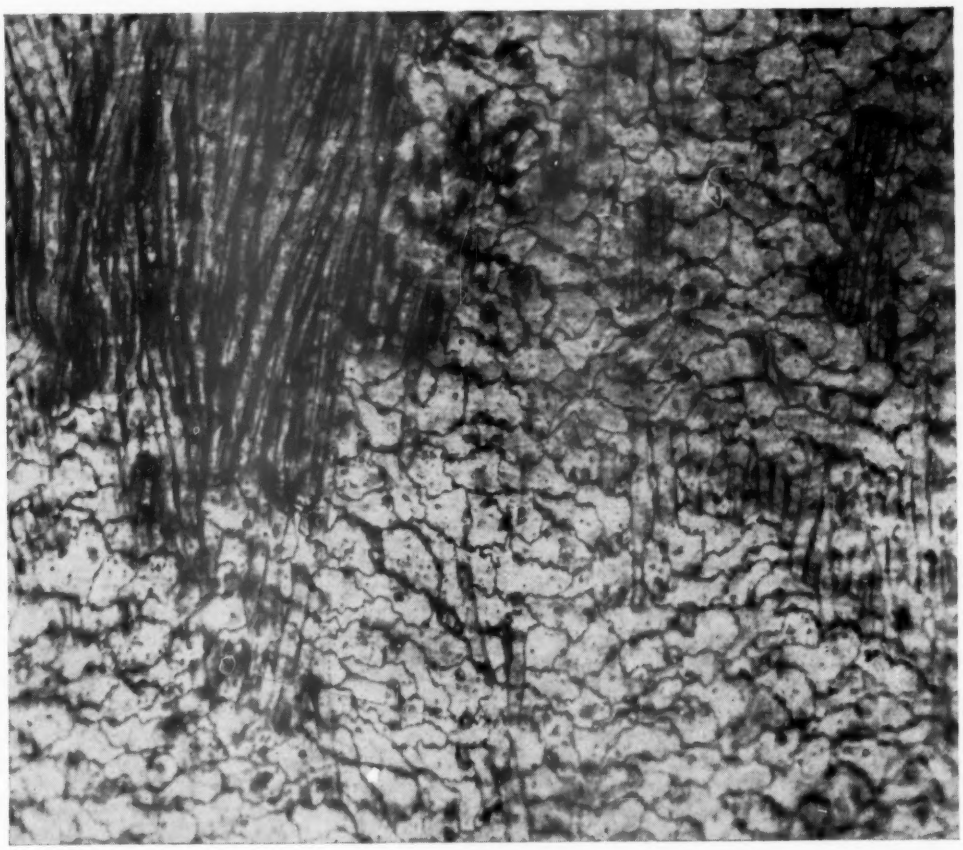

Fig. 3.—Changes in the endothelial cells after 30 minutes' exposure to hypertonic salt solution (silver nitrate method).

of a pulmonary embolus. In these cases the reaction in the wall of the vein is negligible, and from a histologic standpoint the cells appear normal. Since embolism is most common in these patients, I have suggested the term "emboligenic thrombosis" for this variety of venous thrombosis.

However, if the clot retracts only slightly, the subsequent changes in the clot and in the wall of the vein are very different. The clot has a tendency to attach itself to the wall of the vein which by now exhibits marked vasospasm, so clearly demonstrated by René Leriche. The lumen of the vein, therefore, remains occluded and organization of the clot begins to take place. Inflammatory changes appear in the wall of the vein and these give the microscopic changes characteristic of thrombophlebitis. I believe, however, that these

inflammatory changes are not primary but secondary changes and they produce perivenous irritation and cause marked edema of the extremity due to reflex vasomotor changes. It was because of these changes that my teacher, René Leriche, introduced Novocainization of the regional sympathetic ganglia as an active means of treatment for acute venous thrombosis. Ochsner and De Bakey popularized this method of treatment in this country. Homans showed that considerable lymphatic stasis was taking place in these patients, and practically all observers have shown that the danger of embolism from this variety of venous thrombosis is very low. I have used the term "adhesive thrombosis" to describe this type of reaction of the vein following the intravenous clotting of the blood.

The following studies seem to us to be forceful arguments in favor of this interpretation of the pathologic physiology of venous thrombosis:

1. Phlebographic evidence: After careful injections of radiopaque substances, such as Diodrast, into the peripheral veins of patients suffering from "emboligenic thrombosis," studies show a nonobstructing clot which seems to float in the contents of the vein. On the other hand, patients who have "adhesive thrombosis" of peripheral veins show complete obstruction of the lumen of the involved vein.

2. Anatomic evidence: We have demonstrated at operation such floating clots in patients who had "emboligenic thrombosis." These clots were firm but always attached at one place, and they could be extracted without difficulty. At the time of operation when the veins containing "adhesive thrombosis" are opened, we find that the clot sticks to the wall and can be extracted only with difficulty, usually bit by bit.

3. Therapeutic evidence: We have observed many times the rapid disappearance of edema in patients with venous thrombosis following the proper use of heparin. This change has been so striking in some patients that we were greatly elated, only to have the patient suddenly develop massive pulmonary embolism and die. Several such observations have been published in detail from our clinic and recently Cadenhead and Aberd reported similar cases. My colleague, P. Mandel, became interested in this problem with me, and we developed a precise method of studying the retractility of the clot in patients with venous thrombosis. Our studies definitely show that the retractility of the clot differs considerably in the two clinical forms of venous thrombosis, and that heparinization of the patient tends to change "adhesive thrombosis" into "emboligenic thrombosis." We have studied patients with classical phlegmasia alba dolens who were treated intensively with heparin, and although the edema subsided rapidly, the clots within the vein were found to be very soft, easily fragmented, and simple to extract from the vein. We believe this is evidence of incomplete lysis of the clot, and I therefore believe it may sometimes become dangerous to treat patients with "adhesive thrombosis" with heparin.

From all our studies, I am definitely convinced that the two clinical varieties of venous thrombosis are essentially the same from the onset, and the

pathologic changes which occur within the vein are the result of alterations in the retractility of the clot, over which we have very little control at the present time.

I should like now to say a few things about the localization of the primary source of venous thrombosis. As a result of the work of Doeneke, Payr, Rössle, Neumann, Frykholm, Bauer, and many others, it seems to be the consensus that venous thrombosis usually originates in the veins of the plantar surface of the foot or in the calf muscles, and the progression of the clot proximally is a secondary process.

My assistants and I have made a careful study of this problem, and we have systematically made complete phlebographic studies on all patients with venous thrombosis who came under our care. In this group of patients we have included all the clinical varieties of venous thrombosis which occurred on my own Surgical Service, as well as those patients whom we were asked to study from the Department of Medicine and the Department of Obstetrics. These studies were always instituted as soon as possible after the first signs of venous thrombosis had made their appearance. Many of these patients had extensive venous thrombosis. By our phlebographic studies we were able to localize the site of the venous thrombosis, and we were surprised to find that 60 per cent of the patients showed involvement of the femoral veins, the iliac veins, or the pelvic veins. Ducuing's published reports indicate that he also found about 60 per cent of the patients to have iliofemoral or intrapelvic venous thrombosis. Professor Géry, former Director of the Strasbourg Institute of Pathological Anatomy, had a wide experience in studying autopsy material of this type, and he concluded that more than 80 per cent of the venous thrombosis began at a point above the popliteal vein. It is, of course, possible that this high incidence of iliofemoral and intrapelvic type of venous thrombosis is peculiar to our particular region of France, but on the basis of our studies, we are obligated to take these factors into consideration in outlining a therapeutic regimen.

BIOCHEMISTRY OF VENOUS THROMBOSIS

There seems to be little doubt that alterations in the process of blood coagulation do play a vital part in the production of venous thrombosis. Many attempts have been made to study the various aspects of the process of blood coagulation, but in spite of these studies, we still have much to learn.

I have been impressed by the fact that the factors which bring about hypercoagulability of the blood do not appear to be the same in all patients. For many years my assistants and I have made routine studies of blood coagulation on patients with venous thrombosis. We have grouped these tests under the term "Thrombograms." These studies include a careful analysis of all the factors which are known today to have an influence upon blood coagulation. Because of lack of time, I can only summarize our main findings as follows:

A. *Hyperprothrombinemia* does not always explain the occurrence of venous thombosis. Only 42 per cent of all patients with *thrombophlebitis* showed at the

onset of their illness or prior to any treatment a prothrombin level greater than 120 per cent of normal. Only 55 per cent of the patients with *spontaneous venous thrombosis* showed an increase in the prothrombin level of the blood; while only 28 per cent of the patients who develop *venous thrombosis in the postoperative period* showed a great increase in prothrombin content of the blood. Nearly 25 per cent of patients suffering from *phlegmasia alba dolens* showed from the onset of their venous thrombosis a prothrombin level of less than 80 per cent; and 50 per cent of the patients with the clinical form of *phlebothrombosis* showed hyperprothrombinemia.

Degradation of thrombin by the methods of Lenggenhager or the simpler method devised by Mandel showed that the thrombin which was found was destroyed very slowly in 46 per cent of the patients with *spontaneous "adhesive thrombosis,"* 25 per cent of the cases of *postoperative "adhesive thrombosis,"* and 83 per cent of the cases of *"emboligenic thrombosis."*

C. *Thromboplastic activity* showed no constant changes. Sometimes there was an increased activity in some patients, in some it was normal, and in some cases there was very little thromboplastic activity. This varied so greatly that we did not believe there was any statistical value.

D. *Anticoagulant activity of the blood* also varied widely among our patients. It ranged from being very weak to very active, but with no trend which we felt was of statistical value.

E. *Fibrinogen content* as determined by the usual methods also gave us no statistically stable information.

F. *Retractility of the clot.* This we believe is of real significance and may determine the type of peripheral venous thrombosis.

The degree of hypercoagulability of the blood, as we know it at the present time, is not constant in all forms of venous thrombosis, and this hypercoagulability may manifest itself in a variety of ways: sometimes as an increased number of blood platelets; in other cases by hyperprothrombinemia; in others by hyperthromboplastinemia; while in still others, we may see a marked slowing of the degradation of the thrombin or a weak spontaneous anticoagulant activity. Rarely do we find an increase in the fibrinogen content of the blood.

There are many newer factors which are now known to affect the coagulation of blood: the factors V and VI (Owren), factors VII, VIII, IX (Christmas), and factor X (Koller). It is possible that the key to the problem of intravascular coagulation of blood lies among these factors, but up to the present time, we have not been able to find it. When we analyze carefully these postoperative alterations in the process of coagulation of blood, we see that they all tend toward making the blood hypercoagulable and we also know that all the effects of stress produce very much the same changes in these factors. Therefore, we must conclude that this state of hypercoagulability of the blood is much more common than we have been led to believe. Yet in comparison to the frequency of this state of hypercoagulability of the blood, the clinical condition of peripheral venous thrombosis must be considered as rare.

It is therefore obvious that hypercoagulability of the blood constitutes only *one* of the factors, and even though this is important, it is probably not absolutely essential to the actual onset of venous thrombosis.

In spite of all this, our present-day medical therapy of venous thrombosis, both prophylactic and curative, is based almost entirely on the use of anticoagulants. I am mindful of the value of anticoagulant drugs, but I do not believe their use has done much to solve the problem of venous thrombosis and pulmonary embolism.

In Surgical Clinic "A" of the University of Strasbourg, we have a strict regimen for all such patients, and upon arrival at the hospital, they are placed into one of two groups:

1. Those patients who are to undergo some surgical operation which predisposes to the development of venous thrombosis or any individual who has a predisposition to this type of venous disturbance.

2. The other patients are those who have no history of venous disease in the family and who are not likely to be exposed to any procedure which might bring about venous thrombosis.

In the first group, the patients are carefully studied for the possibility of alterations in the mechanism of the blood coagulation, and when necessary, a complete "thrombogram" is made. Blood is taken every day for determination of the platelet count, the prothrombin activity, and the thromboplastic index. When the prothrombin activity is greater than 120 per cent, we immediately give the patients the proper amount of tromexane or phenylindanedione as prophylaxis against venous thrombosis. I do not believe there should be any set dosage of these drugs, since individual susceptibility varies greatly and we are guided entirely by the daily determinations of the prothrombin activity of the peripheral blood. We keep the prothrombin activity about 50 per cent of normal and have found it unnecessary to reduce it further, since this level protects against the accumulation of an excess of prothrombin without risking severe hypoprothrombinemia and hemorrhage into a hollow viscus or into a wound.

If at the same time the thromboplastic index, which normally runs about 3 minutes by the Mandel method, is reduced to 2 minutes or less, we give the patient heparin or one of the heparinoids. These substances are administered by the intravenous route at the rate of 75 or 100 mg. every 4 hours until the coagulation time stays between 12 and 15 minutes. We have found it unwise to lengthen the coagulation time to 20 or 30 minutes, particularly in patients where arterial grafts are being used and the danger of bleeding is great. We have also had a blood clot develop within an arterial graft; this shows that the intravascular thrombosis can take place even in the presence of hypocoagulability of the blood.

In the years in which we have followed this therapeutic regimen to prevent postoperative venous complications, we have been able to reduce the incidence of these complications approximately 90 per cent in my own Surgical Department. We perform an average of 7,000 major operations a year, and during the past four years we did not have a single case of fatal pulmonary embolism until just a few weeks ago when we had two patients die of

fatal pulmonary embolism within a couple of weeks in spite of carefully controlled anticoagulant therapy. In spite of the benefits which we have been able to derive from well-controlled anticoagulant therapy, we occasionally do observe that the intravascular clot within a deep vein continues to extend and causes painful and disabling sequelae in the extremities. It is because of these complications of venous thrombosis that I believe surgical treatment should have a place in the management of this condition.

SURGICAL TREATMENT OF VENOUS THROMBOSIS

There are two real indications for the surgical management of venous thrombosis. Firstly, I believe that patients who have had more than one attack of serious pulmonary embolism run a great risk of having another which might prove fatal. Such a patient must be protected and the interruption of the large veins leading to the heart should be done without hesitation. Secondly, I believe that certain patients with thrombophlebitis of the phlegmasia alba dolens type, who have continued to have edema of the extremity in spite of adequate anticoagulant therapy, should be considered candidates for surgical operation on the veins, even though the original danger of pulmonary embolism was relatively small.

The surgical methods which I believe are indicated in the management of acute venous thrombosis are four in number.

In our clinic we find Novocainization of the regional sympathetic ganglia, as originally described by my former chief, Professor René Leriche, and his associate, J. Kunlin, to be a sound method of treatment. This was first advocated long before the development of anticoagulant therapy, but it still has a distinct place in the management of certain forms of phlegmasia alba dolens. Ochsner and De Bakey introduced this method of treatment into this country, and from all the reports, I gather that it has been used rather widely. There is still some question about the efficacy of this type of treatment, and since the introduction of anticoagulants, fewer and fewer sympathetic blocks are being done. I think it is well known that it may be very dangerous to infiltrate Novocain into the region of sympathetic ganglia in patients who are being treated with anticoagulants. Fatal accidents have been reported by Küss, Delannoy, Lasceaux, and Teinturier.

The other surgical procedures which may be indicated are as follows:

A. *Ligation of Large Peripheral Veins.*—More than 30 years ago, Klapp advocated this method of preventing pulmonary embolism in Germany, but the method did not receive wide acceptance until the studies of John Homans, Arthur Allen, Linton, and Donaldson emphasized the importance of interrupting the direct pathways to the lungs in patients who had peripheral venous thrombosis. Thebaut, Ward Dax, and Owens published work to support this method of treatment.

In our Surgical Clinic in Strasbourg, my own personal experience has been analyzed, and we found that in 60 per cent of the patients who showed postoperative or postpuerperal venous thrombosis the site of the origin of the clot was either in the femoral vein, the iliac vein, or the deep veins of the pelvis. Under these circumstances, the only way we could interrupt the

direct pathway to the lung would be by ligating the inferior vena cava. I have been hesitant about performing such a major procedure as a prophylactic measure against pulmonary embolism, so except in relatively rare cases, we have relied upon intensive medical therapy. If, however, there is a history of repeated attacks of pulmonary embolism, we do not hesitate to recommend and carry out the ligation of the inferior vena cava. Our greatest concern has been the stasis which the interruption of the vena cava produces in the lower extremities and the retrograde thrombosis which frequently takes place and gives rise to the delayed sequelae with which you are all so familiar.

B. *Phlebectomy.*—Phlebectomy means the removal of the entire segment of the obstructed vein, and it might be compared to arteriectomy, which was introduced by Leriche in 1916 for segmental obliteration of peripheral arteries. It is our belief that phlebectomy is better than simple ligation to prevent the extension of the clot proximally. Actual excision of a part of the vein reduces the vasomotor reaction by eliminating the inflamed vein. We believe this is the point of origin of many of the vasomotor reflexes which are so closely woven into the "pathogenesis" of the postthrombotic syndrome.

C. *Thrombectomy.*—On the other hand, thrombectomy tends to rid the vein of its clot and attempts to restore the venous circulation to its previous state. In our Clinic when we were able to localize the site of the obliteration of a vein, the venous segment proximal to the diseased portion is isolated and temporarily occluded; then a short longitudinal incision is made in the vein and the clot is permitted to exude through the opening in the vein. Gentle suction or the application of smooth forceps to the clot is necessary occasionally to free it from the vein completely. Thrombectomy is only complete when both the proximal and distal ends of the vein bleed freely. Arthur Allen removes the thrombus from the proximal end of the vein and leaves in place the distal portion which is densely adherent to the vein. After the vein wall has been carefully closed with fine sutures, anticoagulant therapy is instituted to avoid the recurrence of venous thrombosis.

RESULTS OF SURGICAL TREATMENT OF ACUTE VENOUS THROMBOSIS

In this group we have excluded superficial venous thrombosis in preexisting varicose veins and have tabulated only the results in patients with deep venous thrombosis who were subjected to surgical management on our Surgical Service at the University of Strasbourg.

Operations performed

A. Acute iliofemoral thrombosis	
1. Ligation of the inferior vena cava	4
2. Phlebectomy (iliofemoral region)	21
3. Thrombectomy (iliofemoral region)	26
Number of cases	51
B. Blue phlebitis (phlebitis with arteriospasm)	
1. Phlebectomy in all 7 cases	
Number of cases	7
C. Thrombophlebitis of the upper extremity	
1. Phlebectomy (axillary vein)	9
2. Thrombectomy (axillary vein)	4
Number of cases	13

Results of This Surgical Treatment.—We believe the results should be evaluated from the standpoint of *pulmonary embolism* and from the standpoint of *latent sequelae.*

A. Our incidence of pulmonary embolism: None of the 71 patients of this group showed evidence of pulmonary embolism, even though all were operated upon in the acute phase of the process and the majority were operated upon as surgical emergencies.

Of these patients, 51 had had one or more attacks of pulmonary embolism just prior to operation and, in the other cases, we feared that massive pulmonary embolism might take place at any moment.

Blood clots from 20 to 30 cm. in length, and even longer, were extracted from the affected vein when the thrombectomy was performed. Massive pulmonary embolism did not occur in any of our cases after the blood clot was removed by thrombectomy. We do not imply, however, that without thrombectomy massive pulmonary embolism would have taken place in all or most of these patients.

We do not believe that a surgeon should hesitate to perform a thrombectomy or phlebectomy if there is danger of massive pulmonary embolism.

By these direct attacks upon the process of venous thrombosis in the peripheral veins, we can further reduce the need for pulmonary vein embolectomy, an operation which even under the most ideal conditions offers only a slight chance for success. I have performed the operation only four times and even though the technical aspects of the operation were satisfactory in all cases, none of the patients survived more than a few hours. Pulmonary embolism is a complication which we should strive to prevent.

We have been impressed by the rapidity with which the vasomotor changes associated with some forms of phlebitis disappear after the clot within the vein has been removed.

B. Latent sequelae of venous thrombosis (treated surgically in the acute phase of the process): We have compared the end results after 2 to 6 years in this group of patients with similar patients who were treated *only* by medical means. In these follow-up studies, we have used the following nomenclature:

1. *Perfect results:* This indicates that there was no trace of the previous venous thrombosis by physical or radiological studies and there was no residual edema.
2. *Slight sequelae:* This indicates that there was no trace of the old venous thrombosis, but there was moderate persistent edema in the affected extremity.
3. *Severe sequelae:* This indicates that there was persistent edema of considerable magnitude with cutaneous sclerosis or the presence of a chronic ulcer on the lower leg.

Analysis of our follow-up studies gave us the following data:

A. 26 patients with iliofemoral venous thrombosis were treated by *medical measures only:*

1. 15 patients were observed from 2 to 6 years
2. 11 patients were observed less than 1 year

Perfect results	9
Slight sequelae	3
Severe sequelae	3

B. 51 patients with iliofemoral venous thrombosis were treated surgically in the following ways:

1. *Ligation of the inferior vena cava*

4 patients were followed over a period of 2 to 6 years

Perfect results	0
Slight sequelae	2
Severe sequelae	2

2. *Phlebectomy in iliofemoral region*

21 patients: 13 followed from 2 to 6 years
8 followed under 1 year

Perfect results	9
Slight sequelae	3
Severe sequelae	1

3. *Thrombectomy in iliofemoral region*

26 patients: 22 followed from 2 to 6 years
4 followed under 1 year

Perfect results	12
Slight sequelae	8
Severe sequelae	2

This group of patients is far too small to permit us to draw conclusions; yet we have the clear impression that patients who have been subjected to thrombectomy or phlebectomy have fewer latent sequelae than similar patients who have been treated medically alone or treated by ligation of the inferior vena cava. On our surgical service only the most extensive types of deep venous thrombosis are subjected to operation, so these figures should be all the more significant. We are now advocating earlier operation in most cases of deep venous thrombosis, with the hope of reducing still more the incidence of the serious sequelae of this process. The long period of disability produced by the severe sequelae constitutes a great economic loss to our patients.

What then can be offered to reduce this disability after it occurs, or when the patients consult the surgeon only after the severe sequelae have been established? In this phase, we see marked *residual edema, cutaneous sclerosis,* and *chronic ulceration* of the skin. We have treated 64 patients with these latent sequelae and 41 have been carefully followed for at least three years after surgical treatment.

In patients with marked *residual edema* who showed evidence of segmental involvement of the deep vein, we have performed complete excision of the diseased portion of the vein and usually a periarterial neurectomy of the accompanying artery.

In patients who had marked *cutaneous sclerosis,* we have performed a lumbar sympathetic ganglionectomy, in addition to the phlebectomy.

In those patients who had *chronic ulceration* of the skin of the lower extremities, we performed the complete excision of the diseased veins below the ulcer and all the veins which were partly or completely obstructed in the vicinity of the ulcer in the manner described by Linton and Gunnar Bauer. The defect was then covered by an autogenous graft of skin, either by a pedicle graft from the opposite extremity or by the method of Lagrot.

All the patients operated upon by these methods were seen regularly for three or more years after operation, and the following is an analysis of 67 patients treated by these measures:

17 of the patients had marked residual edema only
2 of the patients had marked cutaneous sclerosis
48 of the patients had chronic ulcers

	RESIDUAL EDEMA	CUTANEOUS SCLEROSIS	CHRONIC ULCERS
Perfect results	42.9%	100.0%	24.0%
Slight sequelae	21.4%	--	56.0%
Severe sequelae	35.7%	--	20.0%

These figures, even though they are not statistically stable, tend to emphasize to us how difficult and disappointing the treatment of the latent postthrombotic sequelae can be, and all too often recurrences of the pain or ulceration may even cause total disability.

PATHOGENESIS OF THE LATENT SEQUELAE OF VENOUS THROMBOSIS

After many years' study of patients of this variety, I believe that the pathogenesis of postthrombotic edema not only centers around the effects of venous stasis and lymphatic obstruction, as described by Homans, but is also associated with neurocirculatory phenomena, as described by Leriche. Such a concept of the origin of this type of edema could explain the failure of phlebectomy in certain of these cases, whether we perform the operation upon the popliteal vein (as advocated by Gunnar Bauer), upon the femoral and saphenous veins (as advocated by Linton), or even the radical excision of the thrombosed veins (as advocated in our Clinic). In patients with cutaneous sclerosis of this type, I have regularly observed extensive occlusion of the smaller veins in addition to changes in the deep veins at the site of the thrombosis, and I believe that the extension of thrombosis in these small veins is largely responsible for the changes in the skin. In regard to chronic ulcers of the lower leg, whether they are simply associated with varicose veins or with deep venous thrombosis, I have always been able to demonstrate several large and thickened veins immediately under the ulcerated area. These involved veins were never completely obstructed but showed a marked degree of endophlebitis with considerable phlebosclerosis. We have confirmed the observation of Linton that when only sclerosis of the skin is present, the more superficial veins are involved; while in those patients with chronic ulcers, there is considerable involvement of the larger subcutaneous collecting veins. Throughout the ulcerated area, the capillaries are greatly narrowed or organically obstructed.

These alterations in the tissue led Professor C. Kayser, my assistant, Dr. Riveaux, and me to study the oxygen content of the blood at the site of chronic

ulcers. We obtained blood samples from various areas about the ulcer and determined the rate of oxygenation. We made these studies on 95 patients and the following results were obtained:

Average Oxygen Values of Peripheral Blood

Blood from the anticubital vein	8.2 vol. %
Blood from the superficial femoral vein	10.8 vol. %
Blood from a vein of a normal lower extremity	11.8 vol. %
Blood from a varicose vein	12.8 vol. %
Blood from a vein draining the ulcer site	13.1 vol. %
Blood from an artery	16.0 vol. %

We therefore found that venous blood from the upper extremity showed more oxygen utilization than that from the lower extremity. In patients with

Fig. 4.—Roentgenogram taken 10 minutes after the injection of Diodrast into the femoral artery in a case of postthrombotic ulceration of the leg. The superficial veins filled immediately after the injection of the radiopaque substance into the artery.

varicose veins, the residual oxygen content of venous blood is still greater (12.8 vol. %), and blood from the ulcerated area shows a still greater content of oxygen (13.1 vol. %), which means less deoxygenation. These studies led

us to think of the possibility that ulcers of the leg are essentially the result of tissue anoxia. We therefore made special studies upon excised portions of the skin from the neighborhood of the ulcer, and we found that the oxygen consumption, as determined by Warburg's apparatus, was two and a half times greater than the oxygen consumption of normal skin. This makes it necessary to explain local tissue anoxia in the presence of increased oxygen content of the venous blood from that area. This paradox naturally led us to the thought that there are direct communications between the small veins and the small arteries in these areas. Ever since the original report of Sucquet in 1860 and the numerous reports that followed by Hoyer, Eckner, Cadiat, Labbé, and Grösser, we have known that such communications normally exist in the human extremity. It was not, however, until the work of Masson in 1924 that we began to recognize the full significance of these communications, which he called "glomi-neuro-myo-arterials." Much work along this line was carried out, particularly in Germany, by Clara, Spanner, Rother, and Sunder-Plassmann.

Fig. 5.—Peripheral arteriovenous shunts in a patient with congenital varicose veins.

During the past few years, my assistants and I have been interested in making serial arteriograms in patients with ulcers of the leg and certain forms of congenital varicose veins, and we have been impressed by the fact that frequently within ten seconds after the Diodrast is injected into the femoral artery, the veins of the leg and foot fill. Under normal conditions and in patients with obliterative disease in the peripheral arteries, this venous pattern does not appear in less than 20 seconds.

In patients with chronic ulcers, the veins immediately beneath the ulcer often fill first. We have also been able to see strange patterns of tortuous small vessels, which appear to have a large head much like a tadpole. These have been observed most frequently on the plantar surface of the foot. We have excised several of these areas and have studied them under the microscope and we find that they are arteriovenous channels. It appears to us, therefore, that when there is an increased oxygen content of the venous blood, this is the result of the opening of these arteriovenous shunts. {The sclerosis of the tissues, which results from venous and lymphatic stasis, actually strangles the capillaries and makes them unable to receive blood in the usual manner; consequently, this blood is shunted into the venules by means of these Sucquet canals. This could explain the increased oxygenation of the blood in the efferent veins, but at the same time, account for tissue anoxia.}

Some years ago Pratt made studies of 249 patients with varicose veins, and he reported that about 25 per cent of these patients showed abnormal arteriovenous communications. Other studies by Piulachs and Vidal-Barraquer of Spain and by Malan in Italy showed the same finding.

It is my firm conviction, therefore, that these other accessory factors come into play with the stasis in the deep veins and the local venous pressure to produce the clinical picture of the postthrombotic syndrome. It has been our experience that because of these accessory factors, the more limited phlebectomy (as advocated by Linton and Gunnar Bauer) is too often incomplete and the good results are then only temporary. It is my belief that we should add to these procedures upon the deep venous system the complete resection of the diseased veins to and about the ulcers, remove all the patches of indurated skin, and cover the defect with autogenous grafts of skin.

De Takats pointed out that sympathectomy alone was of little value in such instances, but in our Clinic we have found that sympathectomy may be in some cases a valuable adjuvant in the management of chronic ulcers of the leg.

ECONOMIC ASPECTS OF THE POSTTHROMBOTIC SYNDROME

It is almost impossible to determine accurately the total effect of the disability resulting in the world today from the postthrombotic syndrome. As early as 1928 Merz estimated that, to the community as a whole, the cost of the management of chronic ulcers of the leg was equal to that of the cost of pulmonary tuberculosis at that time. Lockhart-Mummery and Smitham calculated that 5 per cent of the total population of England suffered from chronic ulceration of the extremities, and the estimated cost to the government was about three million pounds each year. In Sweden where there are but six million inhabitants, reports show that each year from 300 to 400 new patients with postthrombotic ulcers apply for treatment. In one survey (Djelaly) 53 per cent of these patients have had to have the ulcers dressed each day, and Sturup estimates that each patient had to spend approximately 25 days in bed each year because of these ulcers. One of the most recent surveys of this problem, made by Santler and Wiedmann in Vienna, showed that in 1953, 0.91 per cent of the members

of the social insurance plan suffered from pulmonary tuberculosis and 0.18 per cent had various manifestations of the varicose vein–thrombotic syndrome. The average hospital stay for this latter group was 19.6 days; while only 14.7 days were necessary for patients suffering from all other forms of cardiac and vascular disturbances.

Because of the economic aspect of this disease syndrome, we must all make every effort to recognize the early phases of this syndrome at a time when medical and surgical therapy is most efficacious. Long periods of disability are demoralizing to the patient and are a great economic loss to society as a whole.

Let us then once more express our deep gratitude for the lessons which John Homans taught us over many years, and particularly for his efforts toward the awakening of new interest in the struggle against this great plague.

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SURGICAL TREATMENT FOR ANEURYSMS OF THE AORTIC SINUSES WITH AORTICOATRIAL FISTULA

Experimental and Clinical Study

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AORTICOATRIAL fistula is a rare cardiac lesion which usually results from rupture of an aneurysm of an aortic sinus of Valsalva into the right atrium. The condition is generally incompatible with long-term survival. The problem presented by a patient with this syndrome has prompted us to review its clinical aspects and to attempt to develop a method of surgical repair.

CASE SUMMARY

A 32-year-old white male surgeon had been healthy and active until July, 1954, when after climbing a long, steep hill, he became very dyspneic and had to sit down to rest. Although there was no precordial pain with this episode, considerable palpitation caused him some anxiety. He recovered rapidly after a few minutes rest and since this time has remained essentially asymptomatic except for occasional episodes of palpitation. A few weeks later, on a routine examination by his physician who had followed him closely for many years, a cardiac murmur was noted. It is quite definite that no murmur had existed previously.

Physical examination revealed an alert, healthy young man with normal color. There was no cyanosis, clubbing, or venous prominence. Lungs were clear. The heart was not enlarged. There was a continuous, high-pitched, whistling murmur audible over the entire precordium and maximal in intensity in the fourth and fifth right intercostal spaces close to the sternum. Heart sounds were otherwise of normal quality, and there was no arrhythmia. Blood pressure was 120/60 and pulse was 90. Liver edge was palpable just below the costal margin. The remainder of the examination showed no abnormalities.

Routine urine and blood examinations were within normal limits and serology was negative. Venous pressure was 115 mm. of water and circulation time (arm to tongue) was 14 seconds. EKG showed sinus tachycardia but was otherwise within normal limits.

Fluoroscopy revealed prominence of the pulmonary vascular markings with increased pulsations of hilar vessels and moderate enlargement of the right atrium and of the right ventricle. A roentgenogram of the chest is shown in Fig. 1.

Cardiac catheterization studies by Dr. R. J. Bing on two occasions have shown evidence of a large left to right shunt at the level of the right atrium. Right atrial pressure was recorded as 14/8, while pressure in the right ventricle was 48/0.

On the basis of the history and findings on physical and laboratory examination, a diagnosis has been made of rupture of a congenital aneurysm of an aortic sinus of Valsalva into the right atrium with the establishment of an aorticoatrial fistula.

During the two years since the episode of acute dyspnea, the patient has been under careful observation by his physician and has remained essentially asymptomatic. He has

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avoided strenuous activity but has carried on an active surgical practice and has played golf regularly. The murmur has persisted with no detectable change and there has been no increase in heart size.

CLINICAL ASPECTS

Aneurysms of the aortic sinuses of Valsalva may be congenital in origin, or they may result from syphilitic or bacterial infection. There is some difference of opinion as to the frequency of congenital aneurysms. According to Taussig,[46] the congenital variety is less common than the syphilitic type. However, Oram and East[37] in a recent review of 23 aneurysms of the aortic sinuses found 4 to be syphilitic, 3 were bacterial in origin, and the rest were congenital aneurysms.

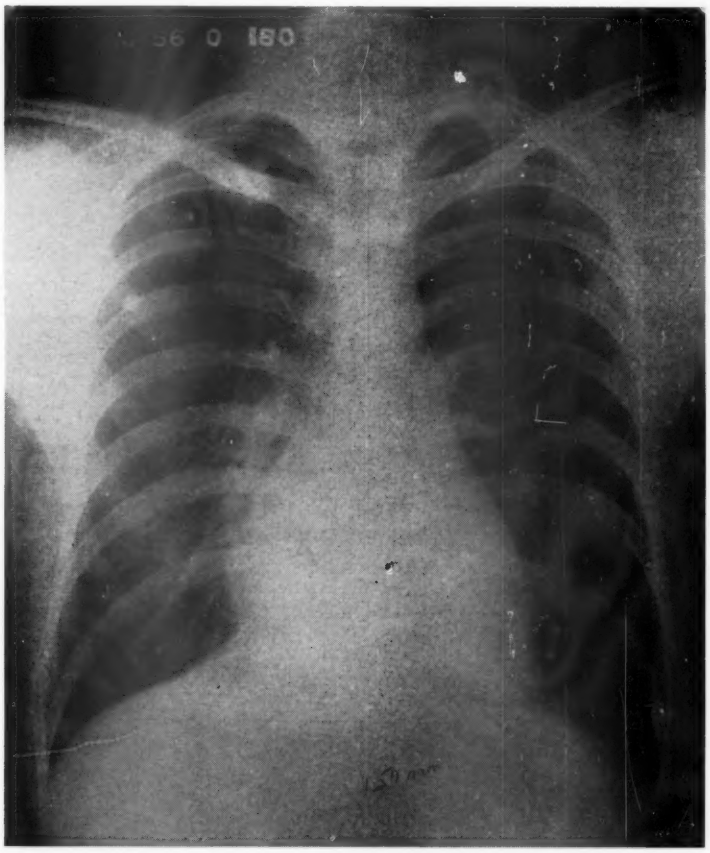

Fig. 1.—Roentgenogram of chest of patient with aorticoatrial fistula 18 months after episode which probably represented time of rupture of aneurysm of aortic sinus of Valsalva into right atrium.

Congenital aneurysms of the aortic sinuses apparently result from a developmental weakness in the aortic wall localized immediately above the annulus fibrosus of the aortic valve. Mall[33] has shown that concomitant with the growth of the aortic septum to divide the primitive truncus arteriosus into aorta and pulmonary artery, the aorta shifts from the right side of the heart to occupy its posterior position opening into the left ventricle. Simultaneously with the shift of the aorta from right to left, the inferior septum at the base

of the ventricles, which is destined to form the upper part of the ventricular septum, shifts from left to right. Eventually, the right margin of the aortic septum fuses with the inferior septum to form the membranous septum. If the aorta reaches its normal position but the fusion between the aortic septum and the ventricular septum is not complete, there results a high interventricular septal defect. Occasionally, the development may progress until the septal fusion is complete, but the aortic wall is attenuated immediately above the annulus fibrosus of the valve in the region of a sinus of Valsalva. With the incessant impact of the pulse wave in the aorta, the weakened base of a sinus of Valsalva develops an aneurysmal sac which may erode through the wall of the adjacent atrium and rupture into the latter, or the sac may erode into the right ventricle just below the attachment of the septal leaflet of the tricuspid valve. Other sites of rupture of sinus aneurysms such as pericardium or pulmonary artery are much less common.

In Fig. 2 is seen a photograph of the region of the root of the aorta of a normal heart in sagittal section at the plane of the posterior or noncoronary aortic sinus and cusp. The close association of the aortic sinus and the right atrium is evident as well as the intimate relationship of the sinus to the membranous septum and the attachment of the septal leaflet of the tricuspid valve. In the same figure, a similar section is shown of a heart in which a congenital aneurysm of the right aortic sinus had ruptured into the right atrium. Dr. Jesse Edwards,[11] to whom we are indebted for these sections, has pointed out that the essential pathologic factor in the congenital aneurysm of an aortic sinus of Valsalva is the separation of the media or elastic layer of the aortic wall from the annulus fibrosus.

In order to assemble data concerning the pathologic characteristics and clinical behavior of congenital aneurysms of the aortic sinuses of Valsalva, a review has been made of 47 cases. This represents all cases which we have been able to find since Thurman's description of the first case in 1840.[1-10, 12-19, 21, 23, 26-29, 31, 32, 34-43, 47-53] The nature of the lesion was confirmed at autopsy in 45 of these cases, while in 2 patients who are still alive the diagnosis rests on clinical grounds with substantiation by catheterization studies. Syphilitic and mycotic aneurysms were not included in this review.

Among the 45 cases confirmed at autopsy, a total of 49 aneurysms of the aortic sinuses were encountered. The aneurysm arose from the right coronary sinus in 34 instances, from the noncoronary sinus in 13, and from the left coronary sinus in 2 cases.*

Rupture of the aneurysm had occurred in 37 of the 45 autopsied cases (Fig. 3). Twenty-six, or 70 per cent, of the ruptured aneurysms arose from the right coronary sinus, and the remaining 11, or 30 per cent, had origin in the noncoronary sinus. Rupture had not occurred in either of the two aneurysms of the left coronary sinus.

*There has been some confusion regarding the nomenclature of the aortic sinuses of Valsalva. In this report, the terms right coronary sinus and left coronary sinus are used in preference to anterior sinus and left posterior sinus. Noncoronary sinus is used instead of right posterior or posterior sinus.

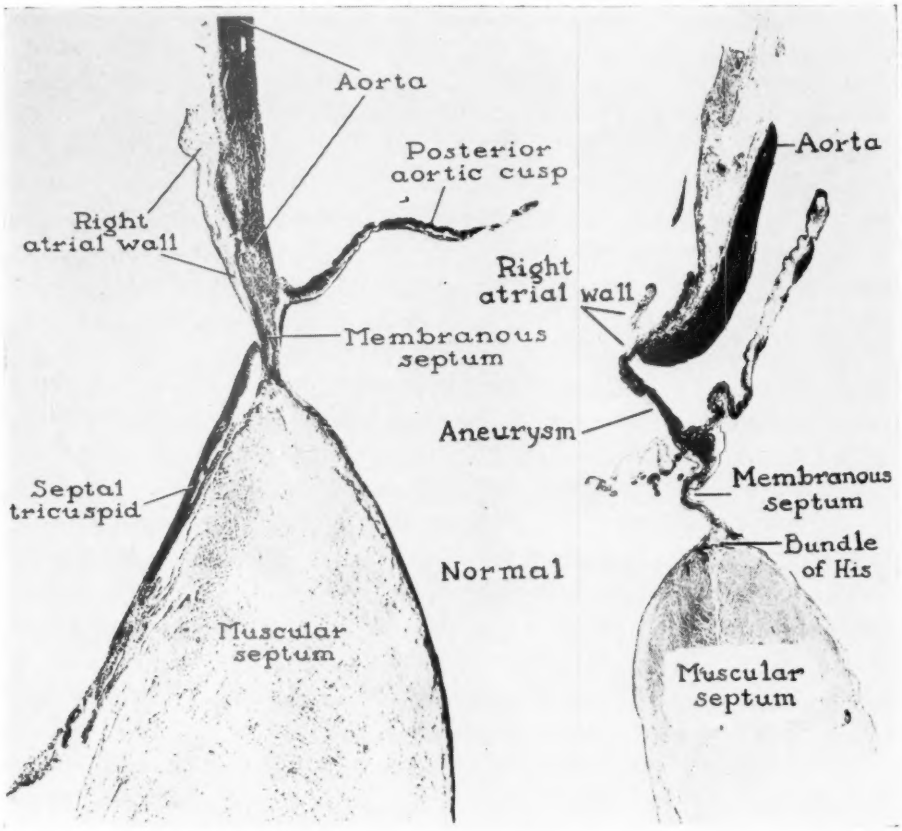

Fig. 2.—Photograph of region of root of aorta of a normal heart in sagittal section at the plane of noncoronary aortic sinus (*A*) compared with similar section of a heart with congenital aneurysm of aortic sinus of Valsalva with rupture into right atrium (*B*). (From Edwards and Burchell, Proc. Staff Meet., Mayo Clin. **31**: 407, 1956.

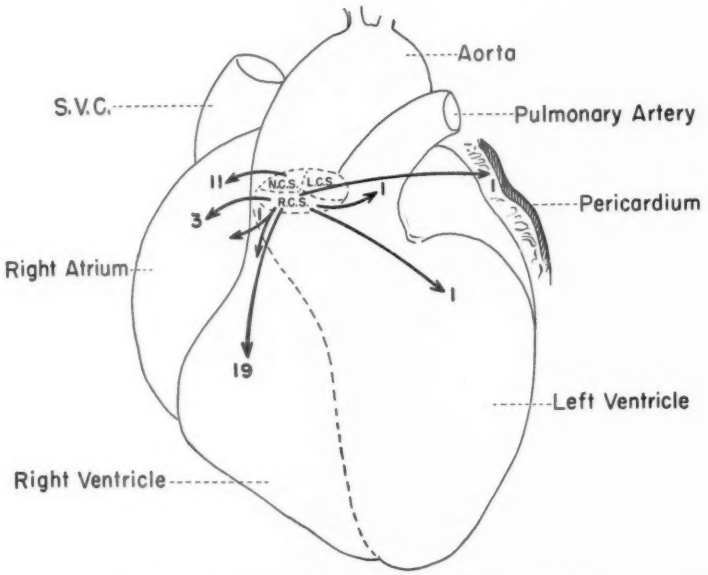

Fig. 3.—Sketch showing sites of origin and terminus of 37 ruptured congenital aneurysms of aortic sinuses of Valsalva confirmed at autopsy.

Table I indicates the terminus of the ruptured aortic sinus aneurysms as determined at autopsy in 45 cases.

TABLE I. TERMINUS OF ALL RUPTURED ANEURYSMS

ANATOMICAL SITE	NUMBER OF CASES
Right ventricle	19
Right atrium	14
Left ventricle	1
Pulmonary artery	1
Pericardial sac	1
Both RA and RV	1
	37

The 26 ruptured aneurysms which arose in the right coronary sinus had a wide variety of anatomical sites of terminus, although 19, or 70 per cent, ruptured into the right ventricle. In 3 instances, this type of aneurysm eroded into the right atrium. There were isolated cases of rupture into the pericardial sac, the pulmonary artery, the left ventricle, and in 1 instance erosion had taken place into both right atrium and right ventricle. In contrast, each of the 11 cases of congenital aneurysm arising from the noncoronary sinus of Valsalva ruptured into the right atrium with the establishment of an aorticoatrial fistula.

The size of the ostia of the aneurysms at their point of origin in the aorta averaged 1 to 2 cm. in diameter with a range of 0.4 to 3.6 cm. In most instances, sinus aneurysms which ruptured into the right atrium fell into a fairly consistent pattern. This consists of a cone-shaped structure which protrudes into the right atrial cavity from the region of the involved aortic sinus. The cone-shaped aneurysm projects inward into the atrial cavity for several millimeters, and a perforation is found at its apex. The apical perforation in the aneurysm has varied from 2 to 8 mm. in diameter. The wall of the sinus aneurysm has been described as thick and fibrous in a few cases but is more often thin and membranous.

Clinical manifestations of congenital aneurysms of the aortic sinuses as reflected by the 47 reported cases are predominantly related to the sequelae of rupture of the aneurysm. These aneurysms occur more frequently in men (82 per cent) than in women. Over 70 per cent of the cases had no symptoms prior to the advent of rupture. While rupture of a sinus aneurysm into the pericardial sac may be immediately fatal, the infinitely more common intracardiac rupture of the aneurysm with the establishment of an aorticoatrial or aorticoventricular fistula is usually not promptly fatal and may be tolerated for months or years.

The syndrome of rupture of a sinus aneurysm into the right side of the heart has a highly distinctive pattern as previously described by Maud Abbott and others.[1, 36] There is usually the sudden onset of substernal or upper abdominal pain which simulates that of myocardial infarction. Radiation into the neck, arms, jaw, or back is, however, unusual. A few patients have had no pain but like our patient have had the sudden onset of extreme dyspnea and palpitation at the time of rupture of the aneurysm. The pain usually lasts several hours, but almost always if the patient survives the immediate

episode, the pain subsides, and the patient enters an almost symptomless phase. This phase usually lasts a few weeks but may be of much shorter or longer duration. Toward the end of this symptomless phase, gross manifestations of an unusual combination of lesions may become evident, namely, those of aortic and tricuspid insufficiency. A strikingly wide pulse pressure with collapsing pulse and systolic expansion of cervical veins with pulsatile liver has been present in many cases. In fact, approximately two-thirds of the reported cases have had manifestations of aortic insufficiency.

The characteristic murmur, which was present in 94 per cent of the reported cases of ruptured sinus aneurysms with intracardiac fistulas, is usually loud, harsh, and superficial in character and commonly accompanied by a coarse thrill. It is usually a shrill, continuous murmur heard best close to the sternum in the third and fourth interspaces.

As a rule, death results from congestive heart failure, the onset of which is apt to be very sudden. The second most common cause of death is bacterial endocarditis. The average age of death in all the reported cases of aortic sinus aneurysms was 33.5 years of age; while in the cases with rupture of an aneurysm, death occurred at an average age of 34.9 years. Rupture of an aortic sinus aneurysm occurred at an average age of 31.2 years. Survival after rupture has ranged from immediate death up to 15 years in 1 case. The mean survival time in the reported cases was 3.9 years, but if 2 patients who lived 10 and 15 years, respectively, are excluded, the average life expectancy is nearer 1 year.

EXPERIMENTAL STUDY

Production of an aorticoatrial fistula in the experimental animal can be readily done by creating a suture anastomosis between the ascending aorta and the right atrial appendage. From such a preparation, information concerning the altered hemodynamics can be obtained, especially as regards the animal's tolerance to fistulas of varying size.

In a series of 14 mongrel dogs, fistulas varying from 3 to 10 mm. in diameter were made by anastomosis between the right atrial appendage and ascending aorta. Cardiac catheterization studies were carried out before operation in each instance and at varying intervals after operation. Data from these studies are summarized in Table II. In general, dogs with fistulas 3 to 4 mm. in diameter tolerated the shunt fairly well, although there was a tendency for right atrial pressure to rise several fold over preoperative levels prior to death several weeks or months after establishment of the fistula. Animals with fistulas 5 to 10 mm. in size tolerated the shunt poorly and, with one exception, died with right heart failure within 10 days after operation.

Production of a Fistula Between the Noncoronary Sinus of Valsalva and the Right Atrial Cavity.—While a suture anastomosis between the aorta and the right atrial appendage serves as a satisfactory means of producing a fistula for physiologic studies, it does not simulate anatomically aorticoatrial fistula as seen clinically. Accordingly, efforts were directed toward producing an actual intracardiac communication between the noncoronary sinus of Valsalva and the right atrium in survival experiments.

TABLE II. SUMMARY OF PHYSIOLOGIC DATA IN DOGS WITH FISTULAS OF VARIOUS DIAMETERS CREATED BETWEEN AORTA AND RIGHT ATRIUM

DOG NO.	DIAMETER OF FISTULA (MM.)	SURVIVAL TIME (DAYS)	CARDIAC CATHETERIZATION	PRESSURE CHANGES (MM./Hg) AT CATHETERIZATION		
				RIGHT ATRIUM	RIGHT VENTRICLE	PULMONARY ARTERY
1	3	14	Preop.	5/0	36/0	
			Postop. (P.O. 7)	19/8	34/0-8	25/13
2	3	70	Preop.	17/5	46/0-4	38/17
			Postop. (P.O. 47)	11/3 (6)*	41/0-8	37/15 (25)
3	3	41	Preop.	8/4 (5)	33/0-8	35/16
			Postop. (P.O. 35)	10/0 (4)	41/0-8	38/8 (25)
4	5	8	Preop.	5/2 (3)	30/0-4	23/14 (18)
			Postop. (P.O. 4)	8/1 (5)	34/0-7	33/0 (19)
5	5	3	Preop.	5/1 (3)	39/0-4	37/23 (30)
6	7	22	Preop.	6/0 (2)	30/0-6	28/13 (21)
			Postop. (P.O. 21)	28/0 (10)	85/0	57/10 (46)
7	8	10	Preop.	8/2 (3)	27/0-8	35/5 (18)
			Postop. (P.O. 3)	19/0 (9)	57/0-16	27/12 (18)
8	8	1	Preop.	6/2 (3)	25/08	
9	8	3	Preop.	5/1	36/0	23/13 (19)
10	9	9	Preop.	6/0 (2)	48/0-5	40/29 (34)
			Postop. (P.O. 2)	18/0 (8)	48/0-7	44/12 (28)
11	10	2	Preop.	4/0	32/0	
12	10	1	Preop.	9/2 (3.5)	40/0-6	26/16 (23)
13	3	Sacrificed (9 mo.)	Preop.	7/0 (2)	29/0-6	27/9
			Postop. (P.O. 21)	(22)	42/0-15	44/20 (30)
14	4	Sacrificed (4 mo.)	Preop.	10/2 (5)	46/0	33/19
			Postop. (P.O. 35)	26/5 (16)	63/0	48/27

*Figures in parentheses are mean pressures.

Preliminary efforts to create such a fistula in the dog by passing a pointed knife or cardiotome through the aortic sinus into the right atrium were unsuccessful because of hemorrhage from the aortic root. Examination of the dog's heart shows that the area of fusion between the base of the aorta and the adjacent right atrial wall is extremely limited. Accordingly, a two-stage procedure was devised in which the first stage consists of tacking the wall of the right atrial appendage to the adventitia of the aortic root so as to promote obliteration of the aorticoatrial sulcus. After 3 or 4 weeks, a second stage procedure is carried out using the technique of hypothermia with inflow stasis.[44] The right atrium is opened and a 4 to 5 mm. fistula is made between the atrium and the noncoronary sinus of Valsalva of the adherent aorta. Inspection of the dog's heart shows that the noncoronary sinus of Valsalva can be entered consistently by passing a pointed instrument or cardiotome through the atrial wall into the aorta at a point 0.5 cm. above the center of the septal leaflet of the tricuspid valve and 1 cm. medial to the ostium of the venous coronary sinus. Better results were obtained by making a stab wound at this point and then removing a small button of fused atrial and aortic wall with forceps

and scissors than by attempting to punch this out with a cardiotome. The atrial incision is then closed and inflow stasis released. After producing such a defect, a thrill can be felt in the atrium, and frequently the atrial wall pulsates with the aortic jet.

A fistula produced in this manner is seen in Fig. 4. Seven dogs with induced defects of this type survived with patent fistulas for a period of several weeks and have had the aorticoatrial shunt closed at a third operation. Cardiac catheterization studies of these dogs showed an increase in right atrial pressure corresponding to that seen in the dogs with fistulas of comparable size produced by suture anastomosis.

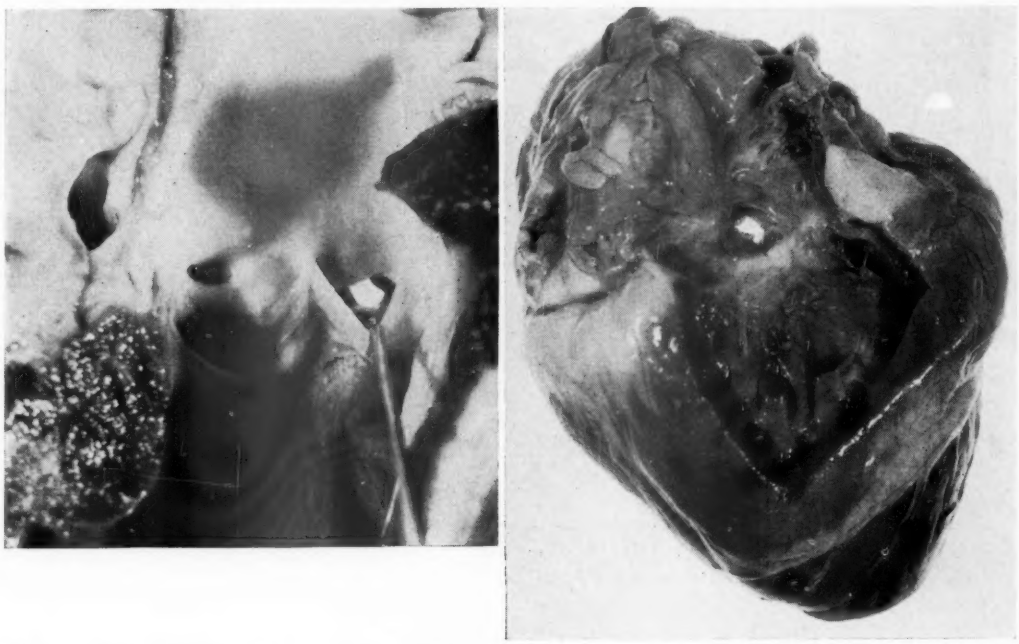

Fig. 4.—Experimental fistula between noncoronary aortic sinus of Valsalva and right atrium: aortic and atrial views.

Closure of an Experimental Fistula Between the Noncoronary Aortic Sinus and the Right Atrial Cavity.—

A. Method with hypothermia and inflow stasis: Fistulas produced as described above have been repaired by direct suture through the open right atrium using the technique of hypothermia with inflow stasis. After preliminary efforts, the following procedure was finally adopted. Hypothermia (of the range 26° to 28° C.) is induced in the anesthetized animal and the thorax opened widely by bilateral anterior intercostal thoracotomy with transection of the sternum at the level of the third intercostal space. The venae cavae are encircled with tapes, the pericardium is opened wide, and the ascending aorta is dissected free from the pulmonary artery and cleared of fatty areolar tissue. A diverticulum is sutured to the ascending aorta as illustrated in Fig. 5. This permits the operator's finger tip to be inserted into the aortic sinus at the site of the fistula. The venae cavae are then occluded and the right atrium is opened.

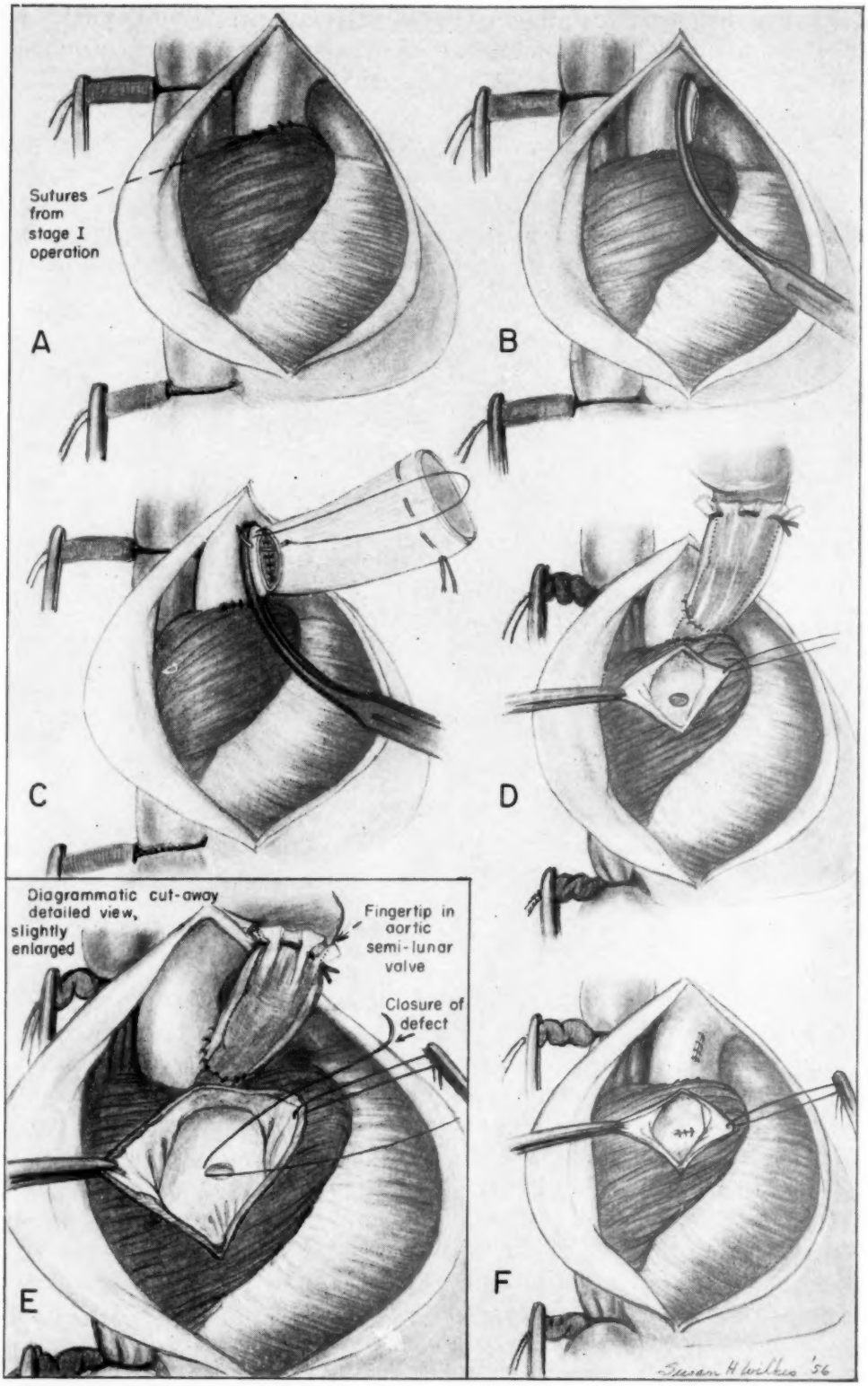

Fig. 5.—Steps in technique of repair of experimental aorticoatrial fistula using hypothermia and inflow stasis.

The finger tip tamponades aortic blood flow through the fistula and protects the aortic valve leaflet from incorporation in sutures during closure of the fistula. Interrupted sutures are then placed in a transverse manner to close the fistula, care being taken to include a grasp of aortic wall as well as atrial wall in each stitch. The finger tip is then withdrawn from the diverticulum whose extremity is occluded. The superior caval occlusion is quickly released, and as air is displaced from the filling atrium the edges of the atrial incision are elevated by stay sutures, grasped in a clamp, and closed by sutures while inferior caval occlusion is gradually released. The total period of stasis is 5 to 8 minutes. The diverticulum is then removed after a clamp has been reapplied tangentially to isolate the aortic incision which is then closed by sutures. The pericardial and thoracotomy incisions are then closed in routine fashion and rewarming is begun.

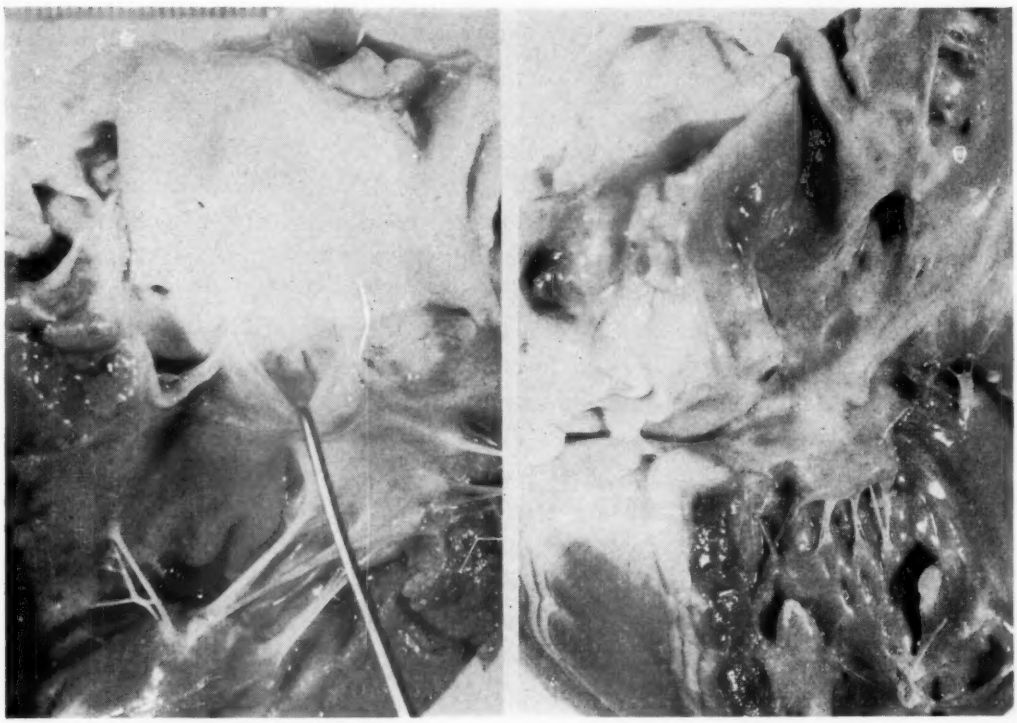

Fig. 6.—Aortic and atrial views of site of surgically closed experimental aorticoatrial fistula 3 months after transatrial repair.

Using this technique or the earlier method without a guiding finger in the aorta, aorticoatrial fistulas have been repaired in a series of 12 dogs, 7 of which had long-standing fistulas, and 5 had production and closure of the fistula carried out simultaneously. Ten dogs survived in good condition 3 or more months before sacrifice, and autopsy showed complete closure of the fistula in each instance with no damage to the aortic leaflet. Fig. 6 shows views of the aortic and atrial aspects of the sutured fistula in one of these animals.

Two other dogs died a few days after operation from acute cardiac failure. Autopsy showed that reopening of the fistula had occurred. In each instance, the sutures had cut through, presumably because of being tied down too tightly.

B. Method with a pump-oxygenator: Until recently, it had been our belief that a technique for closure of an aorticoatrial fistula based on the use of a pump-oxygenator would not be feasible because of the difficulty of obtaining direct visual exposure of the fistula through the atrium while maintaining coronary perfusion. Under these circumstances with venae cavae occluded, blood still enters the atrium from the fistula, the coronary venous sinus, and the thebesian vessels. However, recent experiences in other laboratories and clinics with the combination of a pump-oxygenator and induced cardiac arrest[25] have suggested that such a technique might be used to repair an aorticoatrial fistula. Accordingly, in a small number of animals in which a first-stage obliteration of the aorticoatrial sulcus had been accomplished as described above, preliminary experiments were initiated as follows.

Lightly anesthetized normothermic dogs are submitted to bilateral anterior thoracotomy with transection of the sternum. After heparinization (4 to 5 mg. per kilogram), cannulae are inserted into the superior and inferior vena cavae for the venous pick-up of a DeWall type pump-oxygenator. Arterial cannula is passed into the aortic arch via the left subclavian artery. The pump-oxygenator is connected and adjusted to a flow rate of 30 to 40 ml. per kilogram per minute. Total cardiopulmonary by-pass is then accomplished by occluding the cavae, discontinuing pulmonary ventilation, and cross-clamping the ascending aorta. Cardiac standstill is induced by injecting a few cubic centimeters of a 2.5 per cent solution of potassium citrate in dog's blood into the base of the aorta. With the heart in arrest the right atrium is opened, and a fistula is made as described above between the atrium and the non-coronary aortic sinus of Valsalva. The edges of the aortic defect are then grasped with forceps and elevated; interrupted sutures are placed to close the defect with care to grasp aortic wall as well as atrial wall in each suture and to avoid the aortic valve cusp. After tying and cutting these sutures, the atrial incision is closed almost completely. The clamp is removed from the base of the aorta and the first 30 to 40 c.c. of coronary venous return into the right atrium (containing the potassium citrate) are allowed to flow out of the incompletely closed atrium. After the heartbeat is restored (usually within 30 to 50 seconds) spontaneously, the remaining small defect in the atrial incision is closed with care not to manipulate the heart at this juncture. With satisfactory heart action established, the lungs are reinflated; caval occlusion is released; the pump-oxygenator is disconnected; cannulae are removed; and closure of pericardial and thoracotomy wounds are carried out while an infusion of protamine sulfate (2.5 mg. per kilogram) is started. Citrated blood and sodium bicarbonate solution are infused if needed.

Five dogs have had simultaneous production and closure of an aorticoatrial defect by this technique in a preliminary investigation of the method. Total periods of cardiac arrest have ranged from 10 to 29 minutes. All animals have survived the acute experiment, but four have died in the first few days

after operation. Autopsy has shown a satisfactorily placed and repaired aorticoatrial defect in each instance with no satisfactory explanation for death in these animals as yet elicited by metabolic studies or autopsy findings.

COMMENT

Review of the anatomical characteristics and clinical behavior of congenital aneurysms of the aortic sinuses of Valsalva indicates that these lesions, although uncommon, deserve serious surgical consideration. In common with other types of aneurysms, their inevitable fate is to rupture; but because of their unique anatomical relationships, rupture almost always takes place into the right side of the heart with the establishment of a fistula between the base of the aorta and the right atrium or ventricle. This is most apt to occur in healthy young adults, predominantly men, at an average age of 31 years. The result of rupture of an aortic sinus aneurysm, which is commonly attended by symptoms simulating myocardial infarction, is in effect the formation of an intracardiac arteriovenous fistula. Frequently the presenting clinical manifestations are those of combined aortic and tricuspid insufficiency. Most patients survive the initial effects of rupture but die within an average period of one year with cardiac failure or bacterial endocarditis.

Diagnosis should be suggested by the characteristic clinical syndrome and the finding of the typical high-pitched continuous murmur which is usually maximal one or more interspaces lower than the classical murmur of patent ductus arteriosus. Cardiac catheterization studies confirm the presence of a left-to-right shunt. Retrograde aortography can further demonstrate the precise location and size of the fistula. Review of the reported cases shows that aorticoatrial fistula more commonly (72 per cent) stems from an aneurysm of the noncoronary sinus of Valsalva, and the intra-atrial opening of these fistulas has varied from 2 to 8 mm. in diameter.

The laboratory studies in dogs have shown that experimental aorticoatrial fistulas of 3 to 4 mm. in diameter are fairly well tolerated for many weeks to months while fistulas of 5 to 10 mm. in diameter between the aorta and the right atrium result in death from cardiac failure within a few days in most instances. These findings are similar to those of Scott and Sabiston[45] in dogs with aorticopulmonary fistula where animals with defects larger than 5 mm. in diameter also died within a very few days with failure.

In recent years, there have been a few attempts to close aorticoatrial fistulas in patients. Four such efforts, in each of which a slightly different method was used, have come to our attention. None has been successful and each patient died at or after operation. In one instance, a modification of the method devised by Bailey for closure of atrial septal defects was attempted[24]; and, in another, an effort was made to pass circumferential sutures around the fistula in the aorticoatrial sulcus, but the thrill could never be obliterated.[22] In a third case, a pump-oxygenator was used and the fistula was approached

through the right atrium, but the details of the procedure and problems encountered are not recorded.[37] In the fourth instance in which a transatrial approach under hypothermia was planned, cardiac arrest occurred as the chest was being entered. While an assistant maintained cardiac massage, the operator sutured the aorticoatrial fistula through the open atrium, utilizing a guiding finger passed retrograde down the aorta; but unfortunately, restoration of heart action could not be accomplished.[20]

Review of the reported cases of aorticoatrial fistula examined at autopsy together with the laboratory experience with production and closure of fistulas between the noncoronary sinus of Valsalva and the right atrium clearly indicates that this is an intracardiac lesion which should be repaired under direct vision by carefully placed sutures. As Edwards[11] has pointed out, a sinus aneurysm results from a separation of the elastic layer of the aortic wall from the annulus fibrosus, and it is logical that the repair should be directed at closure of the aortic ostium of the aneurysm by rejoining the superior rim of aortic wall securely to the annulus. While this might theoretically be carried out through the open aorta with the heart in induced arrest, the atrial approach appears to be considerably less hazardous.

The technique of hypothermia with inflow stasis makes it possible to suture experimental aorticoatrial fistula through the atrium with a brief period of stasis and satisfactory survival rates. The short time limit of this method is one of its chief drawbacks. Despite the poor initial survival results obtained in preliminary efforts to produce and close aorticoatrial fistula in dogs by means of a pump-oxygenator and the technique of induced cardiac arrest, this method eliminates the need for extreme haste, which is a distinct advantage. Lillihei[30] has recently accomplished the successful closure of a ruptured aortic sinus aneurysm with a fistula into the right ventricle in a 10-year-old girl by direct suture through the open ventricle using a pump-oxygenator and retrograde perfusion of the coronary sinus. This ingenious use of the pump-oxygenator as well as the combination of the extracorporeal circulation with induced cardiac arrest deserve further evaluation in the development of a satisfactory method for repair of aorticoatrial fistula.

SUMMARY

1. Aorticoatrial fistula results from rupture of an aneurysm of an aortic sinus of Valsalva into the right atrium. An illustrative case is presented.

2. The pathologic characteristics and clinical behavior of congenital aneurysms of the aortic sinuses have been assessed by a review of 47 reported cases.

3. A method of producing a fistula between an aortic sinus and the right atrium is described and studies of a series of animals with this lesion are presented.

4. Experimental methods of surgical repair of aorticoatrial fistula are described.

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Discussion

DR. JULIAN JOHNSON (Philadelphia, Pa.).—I would like to congratulate Dr. Sawyers and his associates on this beautiful piece of work.

We had one of these patients with an aneurysm of the sinus of Valsalva ruptured into the right atrium about 4 or 5 years ago, before we were doing much with hypothermia. This man was in heart failure. A continuous murmur had been discovered 3 or 4 years previously, at which time he had to give up work as a miner.

When he came to us, he was in moderate heart failure. We operated upon him using the atrial "well" described by Dr. Gross. There was a continuous thrill over the right atrium. With the finger in the atrium, a jet was palpable coming through a megaphone-shaped aneurysm, similar to a pulmonary valvular stenosis as felt from above. We were able to take a long clamp and occlude the open end to stop the jet of blood from the aorta. We put a ligature around it. I was afraid that the ligature might roll off. So I decided to persist and placed a triangular suture around the base of the aneurysm. It worked very well except for one fine little jet, which we thought was not enough to justify further sutures.

The patient did remarkably well, and I think he would have been cured for all intents and purposes, except that on his tenth postoperative day he developed an embolus in the superior mesenteric artery. We resected all but the proximal 12 inches of small bowel as

well as the right colon. When autopsy was performed after death due to malnutrition and an intestinal hemorrhage a month later, we found no residual clot in the aorta, but there were parts of the sutures showing through on the aortic side.

I think the method employed here is far superior to our method of repairing the aneurysm.

One other interesting fact is he has a brother who has exactly the same lesion. We catheterized him, but he is hesitant about being operated upon.

DR. DENTON A. COOLEY (Houston, Tex.).—Left-to-right intracardiac shunts may be produced by a variety of anatomic defects and surgeons are now being called upon to recognize many of these anomalies at the operating table. We have had several unique cases of this type in the past month which I would like to describe briefly.

In one patient, a 12-year-old girl, there was a continuous murmur along the right sternal border loudest in the fourth interspace with severe cardiac enlargement. At thoracotomy an enormous right coronary artery measuring almost 2.0 cm. in diameter was discovered. At a point about 6.0 cm. from its aortic end, the coronary widened into a small aneurysm, and a continuous thrill was palpable over an arterial to right ventricular fistula. The fistula was obliterated with several silk sutures. The patient tolerated coronary artery occlusion without change in the electrocardiogram.

In another case, a 31-year-old woman with severe physical incapacity and findings compatible with ventricular septal defect, cardiac catheterization indicated that the left-to-right shunt was at the atrial level. Exploration was done under hypothermia and the defect was found to communicate between the left ventricle and right atrium. Under inflow occlusion the defect was easily repaired through the right atrium. About 8 minutes after the atrium was closed, ventricular fibrillation occurred and attempts to restore cardiac action were unsuccessful. I am confident that this lesion could have been successfully repaired and the patient would have survived if extracorporeal circulation had been used instead of hypothermia.

We have operated successfully upon 2 cases of aorticopulmonary septal defects. In one, a 6-year-old boy with a 2.0 cm. defect, the operation was performed under hypothermia with inflow occlusion. In spite of coronary air embolism and ventricular fibrillation, this boy survived and is well. In another patient 14 years old with a much larger aortic window measuring 3.5 cm. in diameter, the lesion was repaired without technical complication using temporary cardiac by-pass.

Another unusual type of left-to-right vascular shunt occurs in ruptured sinus of Valsalva communicating with the right ventricle. We operated upon an 18-year-old girl with this lesion using cardiac by-pass and the result was very satisfactory. The fistulous opening in the right ventricle was oversewn with silk sutures. At present, by using extracorporeal circulation, almost all types of left-to-right cardiac shunts are surgically correctible except perhaps some of those where the shunt is bidirectional due to secondary pulmonary vascular changes.

DR. LEWIS H. BOSHER, JR. (Richmond, Va.).—In June of 1954, we attempted to repair a ruptured aneurysm of the sinus of Valsalva between the aorta and the right ventricle. The operation was poorly planned. The patient did not survive the operation which was undertaken as a semiemergency.

In the preoperative diagnosis, from catheterization data, it was considered more likely that the patient had a fistula between the aorta and pulmonary artery. At operation the heart was immense and the operative procedure was undertaken with considerable difficulty. A thrill could be palpated in the outflow tract of the right ventricle. The aorticopulmonary artery fistula was absent. The patient's condition was desperate and we felt some attempt must be made to close the fistula. The outflow tract of the right ventricle was opened. The hole was tamponed with the finger while sutures were passed beneath it and tied. The circulation was re-established in 4 minutes, but ventricular fibrillation and failure occurred shortly thereafter.

At post-mortem examination the fistulous tract between the sinus of Valsalva and the right ventricle was found to be 1.5 cm. in length. With only the lower end of the fistulous tract closed, very likely this would have reopened. It seemed wise for future cases either to close the aortic end of the fistula or to tampon the tract with a plug of polyvinyl sponge and suture it from the ventricular side.

DR. FRANK GERBODE (San Francisco, Calif.).—I want to compliment Drs. Sawyers and Scott on their approach to this problem and to report an instance of a traumatic aortico-atrial fistula caused by a bullet. A few years ago, during the Korean conflict, I was presented with a marine who had been shot in the right chest. The bullet passed through the right atrium and aorta, severing an aortic valve and finally becoming lodged in the wall of the left ventricle. He developed severe heart failure. The pulse pressure was wide and a continuous murmur could be heard throughout the base of the heart, giving the impression that a ductus or aorticopulmonary fistula was present. I must confess that we were quite uncertain about the diagnosis. Although I operated, I could not establish the exact location of the communication and the patient subsequently died of heart failure.

At necropsy the fistula was found at the base of the aorta, beneath a severed valve where it communicated with the right atrium. If we had used the right atrial approach as suggested by Drs. Sawyers and Scott, I am quite sure we could have repaired the fistula, but the aortic incompetence would still have seriously impaired the circulation. This lesion could have been diagnosed by cardiac catheterization, and a good repair might have been possible with the aid of extracorporeal circulation.

DR. H. WILLIAM SCOTT, JR. (Nashville, Tenn.).—Certainly the differential diagnosis of this lesion under discussion includes several rather bizarre things, such as coronary arteriovenous fistula and the syndrome of left ventricular-right atrial shunt resulting from a high interventricular septal defect with a defective septal leaflet of the tricuspid valve, both of which were mentioned by Dr. Cooley.

The definitive way to establish the diagnosis of a ruptured aneurysm of a sinus of Valsalva is by the use of contrast roetngenography. This, as yet, has not been done in the patient presented by Dr. Sawyers.

(Slide.) The use of retrograde aortography to demonstrate this type of lesion was accomplished recently in 2 patients by Dr. Glenn Morrow at the National Heart Institute at Bethesda. The first patient, whose film you see here, was a 26-year-old man with clinical and catheterization data compatible with the diagnosis of a ruptured sinus aneurysm. A catheter has been passed via the brachial artery back into the base of the ascending aorta, and with the high pressure injection of contrast material through the catheter, you can see the fistula going from the aneurysm at the base of the aorta down into the right atrium.

(Slide.) Here with the help of the biplane Shaunander apparatus you see the lateral view, demonstrating the fistulous track; probably some portion of this narrow section represents the little cone-shaped aneurysm itself, which tends to project up in the floor of the atrium, as Dr. Johnson mentioned.

(Slide.) In another patient Dr. Morrow recently made the diagnosis in the same way. This is the aortogram of a 20-year-old man with clinical and catheterization findings suggesting a sinus aneurysm with rupture into the right ventricle. Using the same technique as in the first case, a fistulous track was demonstrated passing from the base of the aorta down into the right ventricle.

(Slide.) Here you see rather less well defined in the lateral view the fistula extending from the base of the aorta down into the ventricle.

We hope to get this type of study soon on the patient presented by Dr. Sawyers in order to confirm the diagnosis in a definitive manner.

DR. JOHN L. SAWYERS (closing).—I would like to thank Drs. Johnson, Cooley, Bosher, and Gerbode for their excellent discussion of the paper.

We have delayed operation upon our patient because he has been virtually asymptomatic. However, we feel that from the review of the recorded data on this lesion, he has a dangerous lesion and with development of a satisfactory surgical approach, we contemplate operation on him in the near future.

THE PHYSIOLOGIC EFFECTS OF EXPERIMENTAL TOTAL PULMONARY INSUFFICIENCY

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OPERATIVE treatment for the relief of pulmonic valvular stenosis has been developed to a point where good results are obtainable by blind valvotomy and by open methods employing hypothermia. Occasionally it has become necessary, however, not only to incise and dilate the stenotic valve, but also to remove portions of it before the gradient of pressure between the ventricle and pulmonary artery is eliminated. We have found this to be particularly true in some adults where fibrosis of the valve has caused such a degree of stiffness that satisfactory valvotomy would be impossible without removal of a portion of the valve. In at least two instances, the thickness and fibrosis of the valve membrane were thought to have resulted from a superimposed endocarditis. Perhaps a more common cause is the prolonged effect of turbulence and the jet which is the result of blood passing through a narrowed orifice at high pressure.

Removal of a portion of the usual stenotic membranous obstruction, in our experience, does not result in recognizable pulmonic insufficiency, probably because of the low pulmonary diastolic pressure which is high enough to fold and close the loose cut valve backward toward the ventricle. However, when the valve is fibrotic and stiff there is no such action after partial excision, and regurgitation is inevitable. In two patients this procedure has resulted in frank pulmonary insufficiency with a to-and-fro murmur in the pulmonic area, a right ventricular heave, and cardiac enlargement slightly greater than that which existed when pulmonic stenosis was the only lesion. Although this continues to be of concern, it is noteworthy that both patients can engage in moderate activity and show no evidence of heart failure 2 years after valvotomy and partial valve excision.

In view of these observations in human beings, it was considered advisable to study experimentally the physiologic effect of total pulmonary valve excision, thus causing a free regurgitation.

METHOD

Adult mongrel dogs weighing between 9 and 24 Kg. were used for the study. After preliminary premedication with morphine, they were anesthetized with intravenous Nembutal, 30 mg. per kilogram body weight, together with positive pressure respiration through an endotracheal tube.

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Surgical Procedure.—Pulmonary valvectomy was performed by the method of Kay and Thomas[3] without operative mortality. The chest was entered through the left fourth interspace, and the pericardium opened anteriorly to the left phrenic nerve in order to expose the pulmonary artery and the right heart. Umbilical tapes were placed around the cavae and the azygos vein. In rapid succession the venous return to the heart was obstructed by traction on the tapes, the pulmonary artery was clamped with a rubber-shod clamp at the level of the bifurcation, a longitudinal pulmonary arteriotomy 2 cm. in length was made just distal to the valve ring, and the cusps were excised under direct vision. Immediately following cusp excision, the arteriotomy was closed with a curved Potts clamp, and the circulation re-established. The time of circulatory interruption varied from 1 minute 15 seconds to 3 minutes 25 seconds. The arteriotomy was closed with continuous 5-0 arterial silk.

Cardiac catheterization was performed before valvectomy and again 6 to 10 months after valvectomy in most of the animals. Pressures were measured with a capacitance manometer and recorded with a direct-writing oscillograph. In addition, the cardiac output was measured by collecting the expired air in a Benedict spirometer, measuring the oxygen saturation of the arterial blood and mixed venous blood (from the pulmonary artery) by the Van Slyke technique, and then applying the Fick principle. Four of the dogs were exercised on a treadmill at 6 to 8 miles per hour. Three of these dogs were exercised strenuously for 20 minutes each day, 6 times a week for 8 weeks, but the fourth dog had to be rested after 6 weeks due to pregnancy; she successfully delivered 7 pups 2 weeks later. A chest radiograph was taken before the final catheterization and the dogs were then sacrificed.

Examination of the Heart.—The body weight at necropsy was recorded, the heart removed, and the absence of pulmonary valves confirmed. The pericardium, great vessels, and fat were trimmed away; the heart was washed free of blood, emptied, and then weighed. After fixation in Jory's solution, the right and left ventricles were separated and individually weighed as described by Herrmann.[2] The thickness of the ventricular walls was also measured, taking the mean thickness of the serially sliced ventricles.

RESULTS

Thirteen dogs have been studied. None of the dogs developed right-sided heart failure or an increase in systemic venous pressure. The exercised animals did not become unduly distressed, either immediately after the exercise or at a later date. In all cases, the chest radiograph showed gross cardiac enlargement, mainly on the right side.

Cardiac Catheterization.—Pre- and postoperative studies were carried out on 11 dogs, 4 of which had been exercised (Table I). The right ventricular pressure was increased by 9 mm. Hg or more in 7 animals and was virtually unchanged in 4 (2 of which were exercised). The pulmonary artery pressure was increased in 8 dogs. In 3, an accurate pulmonary artery pressure could not be obtained owing to difficulty in establishing the precise position of the cardiac catheter in the right ventricular outflow tract.

Cardiac Output.—This was measured before and after valvectomy in 8 dogs, 2 of which had been exercised. After valvectomy, cardiac output was increased in 4 (including the 2 exercised dogs) and decreased in 4 (Table I).

Fig. 1.—Typical result of pulmonary valvectomy.

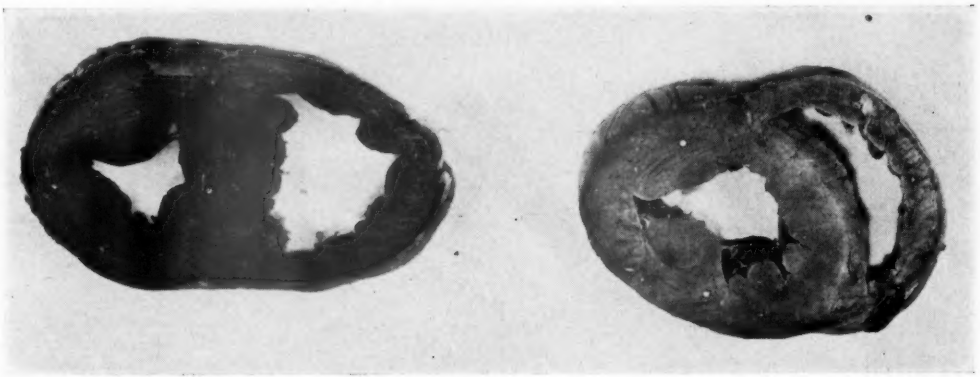

Fig. 2.—Comparison of normal (right) and valvectomized (left) hearts.

Macroscopic Appearance of the Heart.—The pulmonary valve had been excised satisfactorily in all 13 dogs. Some tags of pulmonary valve remained in a few dogs, but these were of no functional significance (Fig. 1). The volume of the right ventricle was increased in all cases, partly due to dilatation (Fig. 2) and partly to hypertrophy (Table II) as shown by the decrease in left-right ventricle ratios.

TABLE I

	DOG NUMBER	CARDIAC OUTPUT (C.C./MIN.)	PRESSURES P.A. (MM./HG)	R.V. (MM./HG)	F.A. (MM./HG)
Preop.	PV4	-	6/0	12/0	-
Postop.		-	20/0	24/0	150/100
Preop.	PV5	-	-	-	-
Postop.		2007	18/0	20/0	160/120
Preop.	PV6	-	-	-	-
Postop.		-	18/0	20/0	160/100
Preop.	PV7	3193	8/2	14/2	170/100
Postop.		1811	22/0	50/0	150/100
Preop.	PV8*	1030	16/8	20/0	170/110
Postop.		-	25/0	25/0	140/110
Preop.	PV9*	1954	18/2	30/0	130/100
Postop.		4071	20/0	30/0	180/130
Preop.	PV10	1805	15/0	20/0	180/130
Postop.		2938	20/0	30/0	160/110
Preop.	PV11*	1612	16/4	26/0	160/140
Postop.		2372	20/5	35/5	170/120
Preop.	PV12	1527	20/5	28/0	180/120
Postop.		1859	30/0	40/5	140/80
Preop.	PV13*	2238	10/4	26/0	160/120
Postop.		-	20/0	35/0	100/?
Preop.	PV14	2229	10/5	25/0	160/120
Postop.		1642	20/0	35/0	150/100
Preop.	PV15	3009	30/4	40/0	180/122
Postop.		1869	32/0	40/0	140/100
Preop.	PV16	1938	18/10	24/0	180/120
Postop.		1344	15/0	25/0	120/70

*Exercised dogs.

TABLE II

DOG NUMBER	MEAN THICKNESS OF VENTRICULAR WALLS (MILLIMETERS) RIGHT VENTRICLE SEPTAL WALL	RIGHT VENTRICLE OUTER WALL	LEFT VENTRICLE SEPTAL WALL	LEFT VENTRICLE OUTER WALL
PV4	5	9	6	13
PV5	6	9	7	15
PV6	8	7	9	16
PV7	8	8	9	15
PV8*	5	8	9	13
PV9*	5	9	7	10
PV10	7	6	6	15
PV11*	7	6	6	12
PV12	5	5	5	14
PV13*	8	8	8	15
PV14	7	8	9	14
PV15	6	8	6	12
PV16	5	9	8	16

*Exercised dogs.

Ventricular Weights.—Herrmann found that the average left ventricle to right ventricle weight ratio in 200 normal dogs was 1.39, with a maximum of 1.77 and a minimum of 1.15. In our series (Table III), the left ventricle to right ventricle weight ratio of 12 dogs was less than Herrmann's average figure of 1.39; and in 10 dogs the ratio was less than Herrmann's minimum figure

TABLE III

DOG NUMBER	WEIGHT IN GRAMS					RATIOS		TIME INTERVAL OPERATION/ SACRIFICE (MONTHS)
	BODY AT SACRIFICE	HEART		RV	LV		HEART WT./ BODY WT.	
		UNFIXED	FIXED	FIXED	FIXED	LV/RV		
PV4	14.1	111.0	102.0	46.5	38.5	0.85	.0079	10
PV5	12.1	100.0	96.5	45.0	36.5	0.81	.0082	10
PV6	24.2	224.0	200.0	65.2	102.8	1.57	.0093	10½
PV7	21.7	179.0	164.0	71.6	70.4	0.98	.0082	9½
PV8*	9.5	93.0	87.3	35.4	40.1	1.13	.0097	9
PV9*	11.5	90.0	84.5	32.1	40.0	1.24	.0078	8
PV10	9.1	87.0	85.0	34.0	41.4	1.22	.0095	7½
PV11*	9.7	90.0	87.5	32.1	34.7	1.08	.0093	7
PV12	10.3	95.0	89.0	37.5	35.0	0.93	.0092	6½
PV13*	14.9	146.0	137.5	60.3	58.0	0.96	.0098	9
PV14	10.0	103.0	93.0	40.5	41.5	1.02	.0103	6
PV15	11.0	99.0	91.2	40.5	37.2	0.92	.0090	6
PV16	14.1	103.5	99.0	40.0	42.5	1.06	.0073	6

*Exercised dogs.

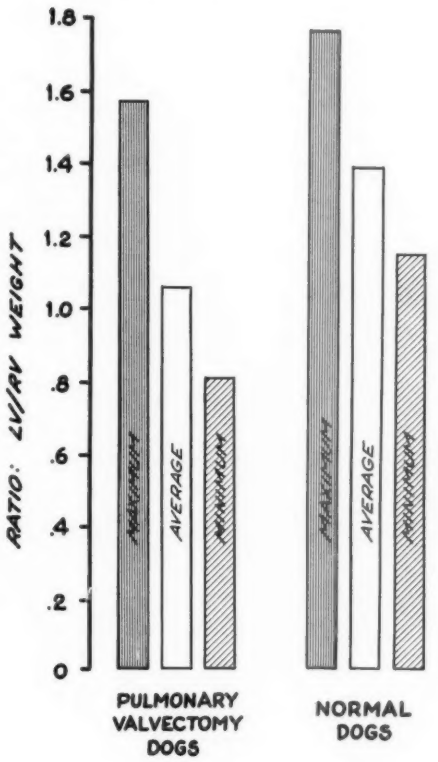

Fig. 3.

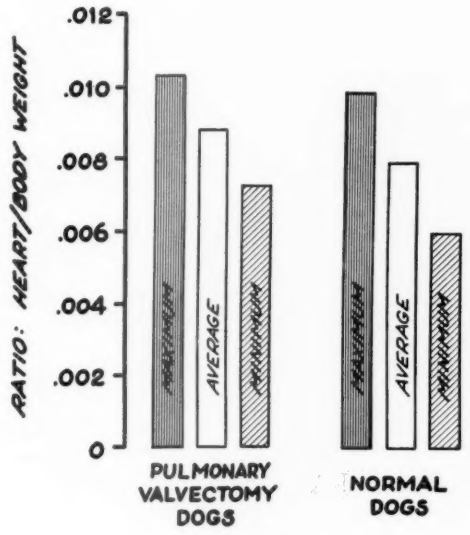

Fig. 4.

Fig. 3.—Ratio of weight of left ventricle to weight of right ventricle.
Fig. 4.—Ratio of heart weight to body weight.

of 1.15; thus indicating that in these 10 dogs, definite right ventricular hypertrophy had occurred (Fig. 3). In addition, the right ventricle was heavier than the left in 6 dogs, which is the reverse of normal. The ratios of the 4 which were exercised were 0.96, 1.08, 1.13, and 1.24.

Comparing the total heart weight to the body weight, Herrmann found that the average normal ratio was 0.00798, with a maximum of 0.00994 and

a minimum of 0.00600. In our series (Fig. 4), 9 dogs were above the average normal ratio but below the maximum, 1 was above the maximum, and 1 below the average but above the minimum. Thus, although in only 1 case could it be stated that there was definite hypertrophy, the trend was toward a figure above that of the average normal.

Ventricular Thickness.—Measurements of the mean thickness of the ventricles are presented in Table II.

DISCUSSION

Several workers[1, 4, 5] have produced varying degrees of pulmonary insufficiency by excision of part of a cusp or dilatation of the valve ring. Neither of these procedures caused congestive heart failure or a reduction in exercise tolerance. Kay and Thomas[3] excised all 3 cusps of the pulmonary valve in 15 dogs, thus producing total insufficiency. Although 1 dog developed right-sided heart failure and died after 8 months and 1 died of inanition, 13 remained well until sacrificed after periods of up to 31 months. They all showed signs of right ventricular dilatation. None of them had been exercised.

In the study reported here, a total insufficiency was produced which resulted in dilatation and hypertrophy of the right ventricle. This hypertrophy was demonstrated by an increase in left-right ventricle ratios in the majority of animals. The right ventricle was heavier than the left in 6 dogs, which is the reverse of the normal. It is of interest that 4 of the animals were able to undertake 20 minutes of strenuous exercise 6 days a week for 2 months without any apparent ill effect, although, of course, it is not known for how long this exercise could have been tolerated. In view of the very definite structural changes that were apparent in the heart after sacrifice of the dogs, it is likely that the extra burden of pulmonary insufficiency would have led to cardiac failure ultimately.

SUMMARY

A study has been made of the physiologic and pathologic changes that occur following complete excision of the pulmonary valve in 13 dogs.

None of the dogs developed heart failure during the period between operation and sacrifice (6 to 10 months). Four dogs were able to undertake regular strenuous exercise for 2 months without signs of heart failure.

The right ventricular pressure and pulmonary artery pressure because increased in two thirds of the animals after valve excision.

The change in cardiac output was equivocal—in 4 dogs, it increased; and in 4 dogs, it decreased.

There was definite hypertrophy of the right ventricle. This was shown by changes in the weight ratios of right/left ventricle, and heart/body weight.

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Discussion

DR. H. WILLIAM SCOTT, JR. (Nashville, Tenn.).—I have enjoyed this presentation by Drs. Ratcliffe, Gerbode and their associates very much and I think it is of considerable importance. We have been interested in this problem of pulmonary insufficiency with particular reference to the tolerance of the heart to the damage inflicted by an incision placed in the wall of the pulmonary artery and carried directly across the annulus of the valve into the base of the right ventricle.

The reason for considering such an incision, as mentioned yesterday by Dr. Lillihei in a panel discussion of open cardiac surgery, is to permit the operator to view and deal with the whole outflow tract of the right ventricle in the presence of combined valvular and infundibular lesion. A few months ago we lost a child with a combined valvular and infundibular lesion largely, I believe, because of our failure to use such an incision. In this patient we exposed the pulmonic valve by incision in the artery with Swan's method of hypothermia and stasis. The valve was repaired and a muscular infundibular diaphragm was disclosed immediately subjacent to the annulus. The arterial incision was occluded and stasis released with the plan in mind of attacking the infundibular diaphragm through a separate ventricular incision below the annulus after a 10 or 15 minute recovery period. Unfortunately, recovery did not take place and the heart went into arrest, almost certainly because the high-grade obstruction had not been totally relieved during the brief anoxic period of stasis.

Recently in our laboratory, Dr. Royce Dawson has been studying the effects of widely opening the outflow track of the right ventricle under hypothermia and stasis by an incision extended from the pulmonary artery across the valve ring into the right ventricle. In some animals the valves have been excised. The widely opened artery, annulus, and ventricular wall are repaired by simple linear closure and the effects of this damage to the valve are assessed by studies similar to those made by Dr. Gerbode and his associates. The results, thus far in observation for only 4 or 5 months, seem to indicate that tolerance to this type of damage is fairly good. We have found nothing in the catheterization data to indicate any significant degree of impairment. These studies obviously will have to be carried on for an extended period of time to determine the long-range effects.

DR. FRANK GERBODE (closing).—This problem occurred to us as being an important one to study because we must frequently remove portions of the pulmonary valve membrane to eliminate the gradient in pulmonic stenosis. Pulmonary insufficiency does not occur if a segment of pliable stenotic membrane is removed. However, we find quite a variation in the pathologic situation presented to a surgeon at these operations. One of them has been mentioned by Dr. Ratcliffe; this is a pathologic change which makes the valve stiff and hard and almost impossible to cut or dilate. This type of valve has to be excised to a certain extent to obtain a satisfactory result. One can excise enough to correct the stenosis without producing physiologically significant insufficiency in most instances; at times more must be removed which results in incompetence.

There is also a condition which I would like to call the "suicidal heart." In this form of stenosis there develops a right ventricular hypertrophy which gradually narrows the outflow tract and produces a sort of infundibular stenosis. This develops from the work entailed in pumping against the stenotic valve. In its advanced form I believe that it probably shuts off the flow from the right ventricle before it is empty, and therefore adds to the burden caused by the already existing valvular stenosis.

In the course of our efforts to relieve this form of infundibular stenosis, or "suicidal" hypertrophy, of the right ventricle in two patients, we removed so much muscle with portions of the valve that considerable insufficiency resulted. Both patients have loud to-and-fro murmurs and large hearts and I am uncertain as to their eventual fate. This form of self-increasing infundibular stenosis may explain some unsatisfactory results from pulmonary valve surgery.

DACRON TUBE AND BIFURCATION ARTERIAL PROSTHESES PRODUCED TO SPECIFICATION

EXPERIMENTAL AND CLINICAL USE

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(From the Departments of Surgery of the University of Illinois and the College of Physicians and Surgeons, Columbia University, New York, and the Surgical Services of Presbyterian Hospital, New York, N. Y., and St. Luke's Hospital, Chicago, Ill.)

MANY types of vascular prostheses have been developed since the demonstration that synthetic materials could be used as arterial substitutes.[4] These have differed in respect to plastic material used, size of yarn, tightness of weaving, knitting or braiding, and in the form and shape of the prosthesis.

In their ultimate development, prostheses may be expected to provide certain advantages over human vascular tissue used as grafts. These may be listed as (1) availability, (2) standardization in the sense of known reliability and limitations, and (3) freedom from problems of sterilization and storage. The factor of availability which will be provided by reliable prostheses is particularly needed in areas where small population or other factors make the obtaining of human tissues unsatisfactory. This availability will increase the application of newer vascular surgical techniques by qualified surgeons throughout the country. Certain disadvantages of synthetic graft materials not easily overcome are (1) the inherent difference in handling characteristic which makes them unfamiliar to the surgeon, accustomed to working with tissues, (2) the fact that such prostheses always represent a foreign body, a point which is of importance largely in the event of accidental contamination, and (3) lack of elasticity. The factor of unfamiliarity may be overcome in the development of materials having flexibility and by practice in handling of available synthetics. The foreign body factor in the presence of infection is probably a disadvantage which is shared by arterial and other homografts, although in part it was responsible for the loss of two prostheses in the clinical cases to be presented. Elasticity would theoretically be a desirable characteristic for arterial prostheses, but it is unlikely at the present that a material will be developed which is at the same time elastic and durable. Elasticity is probably reduced, even in the best homografts after they have been implanted for a time.[1]

The present report is concerned with the results of an evaluation of an improved synthetic prostheses of Dacron fabric. Improvement has been through the development of a weaving method which allows the construction of tubes and bifurcations of any desired size or shape. The seam usually sewed to make

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a prosthesis from woven material has been produced by the weaving process. The method imparts a strength of seam not achieved by the sewing machines and a smoothness of the inner surface greater than that which can be obtained by even the closest mechanical stitching.

METHOD OF PRODUCTION OF PROSTHESES*

The method of weaving resulted in the simultaneous weaving of two layers of Dacron fabric of the taffeta type. The automation guiding the process, which is carried out on a Jacquard loom, is so arranged as to produce a double row of interlocking threads producing the seam outlining any desired pattern. By this process, straight and curved-end tubes of various sizes and bifurcations in a number of sizes have been constructed (Fig. 1). The straight tubes were fabricated in 5 to 10 mm. diameter sizes in increments of 1 mm. The curved-end

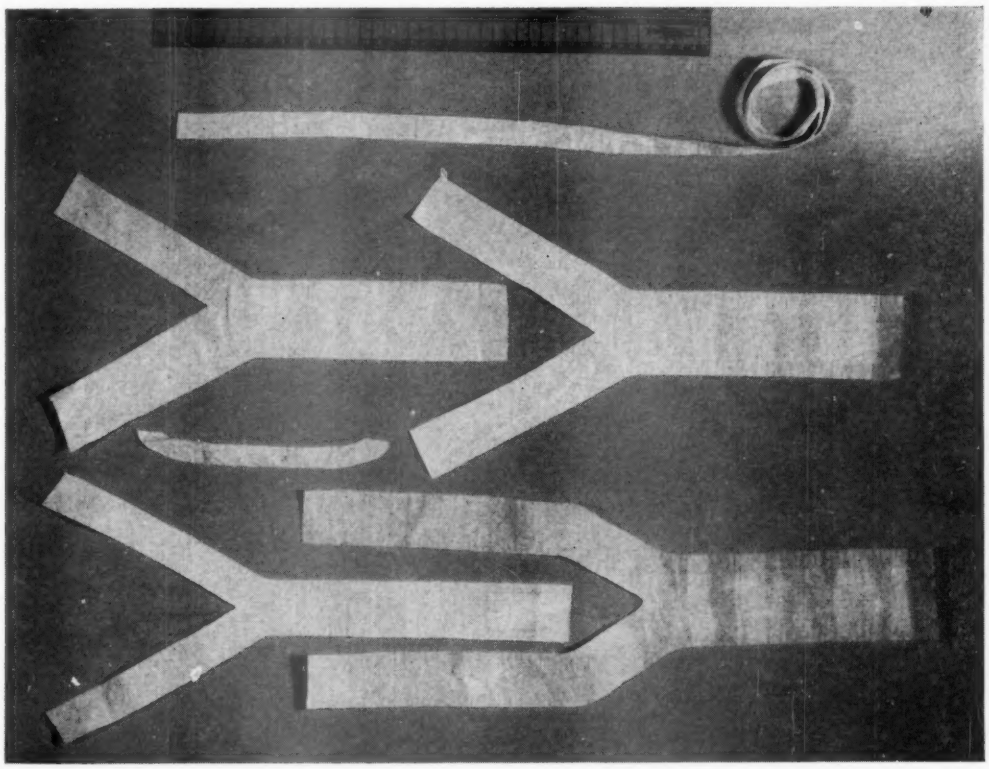


Fig. 1.—Some of the available Dacron prostheses, including a long straight tube, 4 bifurcation patterns, and a 15 cm. curved-end tube.

tubes were made in 8 mm. diameter and in length of 15 and 20 cm. Five sizes of the bifurcation so far have become available. Twelve are planned. The thread count of the fabric is 100 × 150 per inch. The yarn is 70 denier with 32 filaments, having 9 turns to an inch. The resultant fabric is moderately porous.

On the inner surface of the tubular structures, the selvedge type of seam is smooth and continuous with the adjacent material. Separation of the seam with manual pull is virtually impossible since it has the same strength as the fabric.

*Planning and production was with the assistance of Mr. William Liebig.

The finished yardage contains duplications of a desired pattern. These are easily cut out with a heated needle, producing a frayproof edge. The prostheses are washed thoroughly in hot detergent solution and then passed through at least five rinses of water to remove the detergent. We have preferred sterilization by standard autoclaving technique at the time of use.

EXPERIMENTAL DATA

Animal trials of this new material were carried out by two techniques in the laboratories of the departments of surgery of two institutions. Straight tubes 6 to 8 mm. in diameter and from 45 to 103 mm. in length were implanted

Fig. 2.—Aortogram of a dog in which a curved-end tube was implanted from dorsal to lumbar aorta.

by end-to-end technique in abdominal or thoracic aortas of dogs. Proper tension was achieved by resecting a segment of aorta somewhat shorter than the length of the prosthetic replacement. The ends of each tube were prepared by heat fusion. A simple over-and-over suture with 5-0 braided silk was used for the anastomoses.

The second technique utilized the curved-end tubes to form a by-pass extending from the thoracic to the abdominal aorta in dogs traversing the diaphragm (Fig. 2). In these experiments, which are limited to two dogs, the graft ends were similarly prepared by heat fusion and anastomosed end-to-side with an over-and-over suture.

RESULTS

Straight tubes have been implanted in a total of 53 animals of which 21 were in the thoracic aorta (Table I). Of the latter, 16 were done at Columbia University. These tubes, 1 cm. in diameter and 8 to 10 cm. long, have been observed up to one year. Two dogs died of pulmonary infection at 6 and 8 weeks, respectively. The grafts were completely patent. Four of this group have been sacrificed at 3 and 4 months. In three the grafts were entirely patent, while in the fourth a small mural thrombus was present at the distal suture line (Fig. 3). The remaining 10 dogs survive for long-term observation, the grafts all being patent. Patency has been confirmed in 6 of this latter number by recent aortography.

Fig. 3.—Photograph made at autopsy of a Dacron graft which had been in thoracic aorta of a dog for 4 months.

TABLE I. THORACIC STRAIGHT TUBES

	UNIVERSITY OF ILLINOIS	COLUMBIA UNIVERSITY
Number of dogs	5	16
Length (cm.)	5.5 to 7.5	8.0 to 10
Diameter (mm.)	13.7	10
Time (days)	1 to 245	42 to 365
Number patent at autopsy	4*	6*
Number patent at aortogram (survivors)	1	6
Number clinically patent (survivors)	-	4
Per cent patency	100	100

*One in each series showed a small mural thrombus at a suture line.

Five of the 21 thoracic grafts were done at the University of Illinois. These varied in length from 55 to 75 mm. and were 13.7 mm. in diameter. One in this group died at the end of 4 days, showing complete patency of the graft. Three were sacrificed at 14, 14, and 168 days respectively, to make observations on the healing process and patency. Two of these were completely patent while one showed a small mural thrombus at the distal suture line. The fifth dog of this group has been permitted to survive, and aortography at 250 days showed complete patency of the graft.

Thirty-two animals have been subjected to resection of the abdominal aorta and replacement with grafts at the University of Illinois (Table II). These vary in length from 45 to 110 mm. and in diameter from 6 to 10 mm. Four animals which are now being kept for long-term examination were studied by aortogram at 6 and 12 months, demonstrating complete patency of the graft. Five animals which died or were sacrificed 1 to 23 days after implantation showed almost complete or complete thrombosis due to kinking of the graft. The 23 remaining animals sacrificed at intervals up to 287 days showed good patency of the lumen of the graft. In the 2 animals with the 15 cm. by-pass graft between the thoracic and abdominal aorta, patency has been established at 7 and 8 months, respectively (Fig. 2).

TABLE II. ABDOMINAL STRAIGHT TUBES*

Total number of dogs	32
Length of grafts (mm.)	45 to 110
Diameter of grafts (mm.)	6 to 10
Time (days)	1 to 352
Number patent at autopsy	23
Number patent at aortogram (survivors)	4
Number thrombosed	5
Per cent patency	85

*University of Illinois.

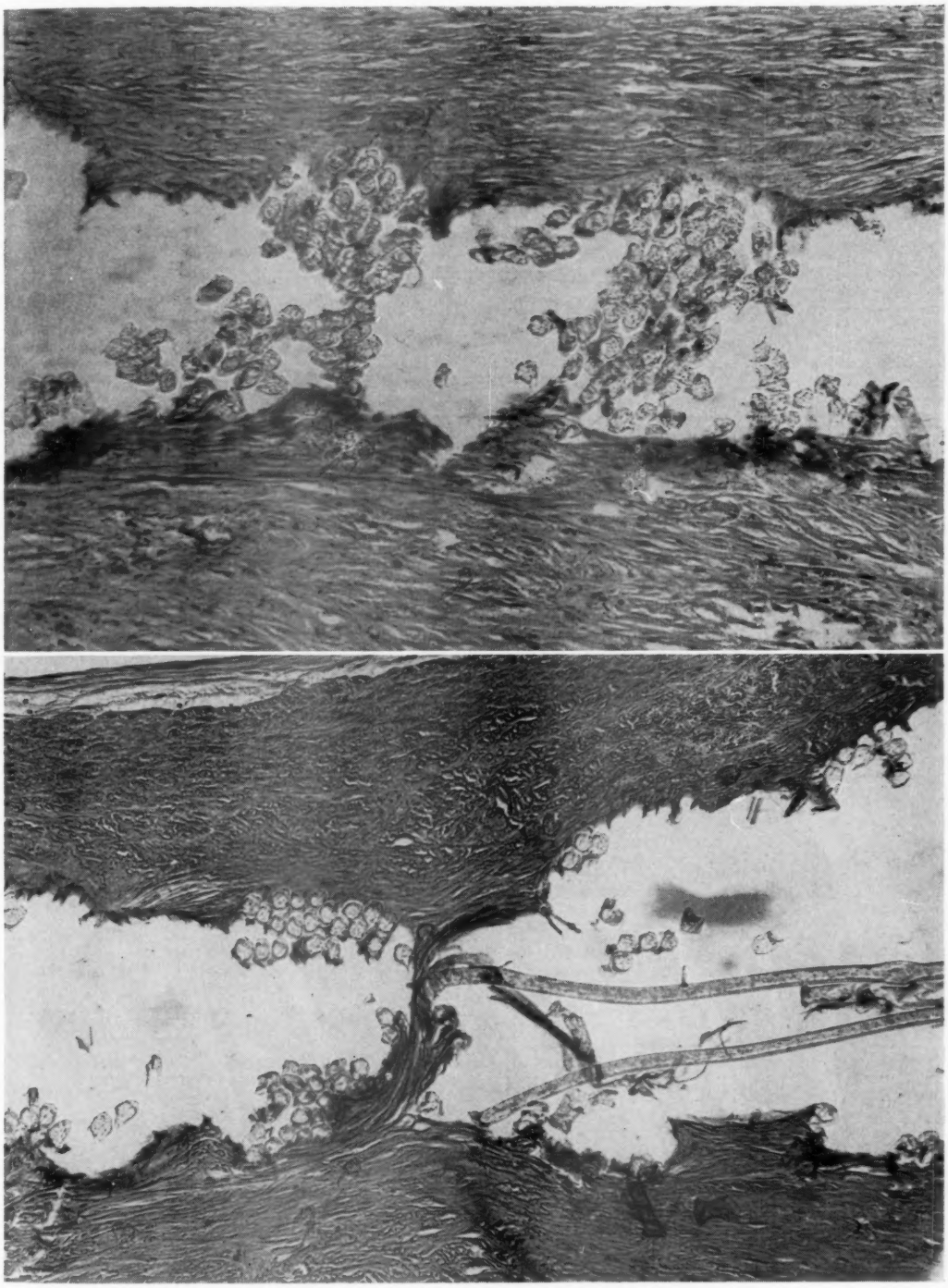
In these animals, occasional mural thrombi were observed, covered by a smooth lining which was continuous with the lining over the surface of the remainder of the fabric. Another deviation from perfectly smooth assimilation of the prosthesis into the arterial channel was the occasional appearance of some degree of kinking of the wall. Explanation of this must be sought as possibly being due to implantation of a tube too long for the defect and therefore under insufficient tension, or to contraction of surrounding scar tissue, or to contracture of hematoma incompletely removed after implantation of the graft. It was noted that the inner surface of the selvedge seam was not the site of mural thrombi in those instances in which they were observed.

Microscopic examination of the grafts revealed integrity of the synthetic fabric. A dense fibrous tissue envelope was present after 8 to 12 weeks. In first 2 to 3 weeks, the surrounding tissue showed an early healing process. The fabric was penetrated by fibroplastic proliferation, connecting the outer fibrous tissue envelope with a much thinner internal lining. The latter was composed of cells similar to the outer sheath. The internal lining consisted of cells not differing from the underlying fibroblasts, but having an orderly cellular appearance. The origin of these cells is not evident (Fig. 4, *A* and *B*).

CLINICAL MATERIAL

Suitable cases of abdominal aortic aneurysm, thrombosis of the aortic bifurcation, and peripheral segmental arterial occlusion were selected for clinical trials of the bifurcation and tubular material. Chronic arterial occlusion in the lower extremities was treated by the implantation of 8, 9, and 10 mm. straight tubes placed as by-pass grafts and 8 mm. curved-end tubes, also installed as by-passes. The segments of occlusion so treated varied in length from those which could be traversed by the 20 cm. curved-end tube, to those which required by-passes extending from the common femoral artery

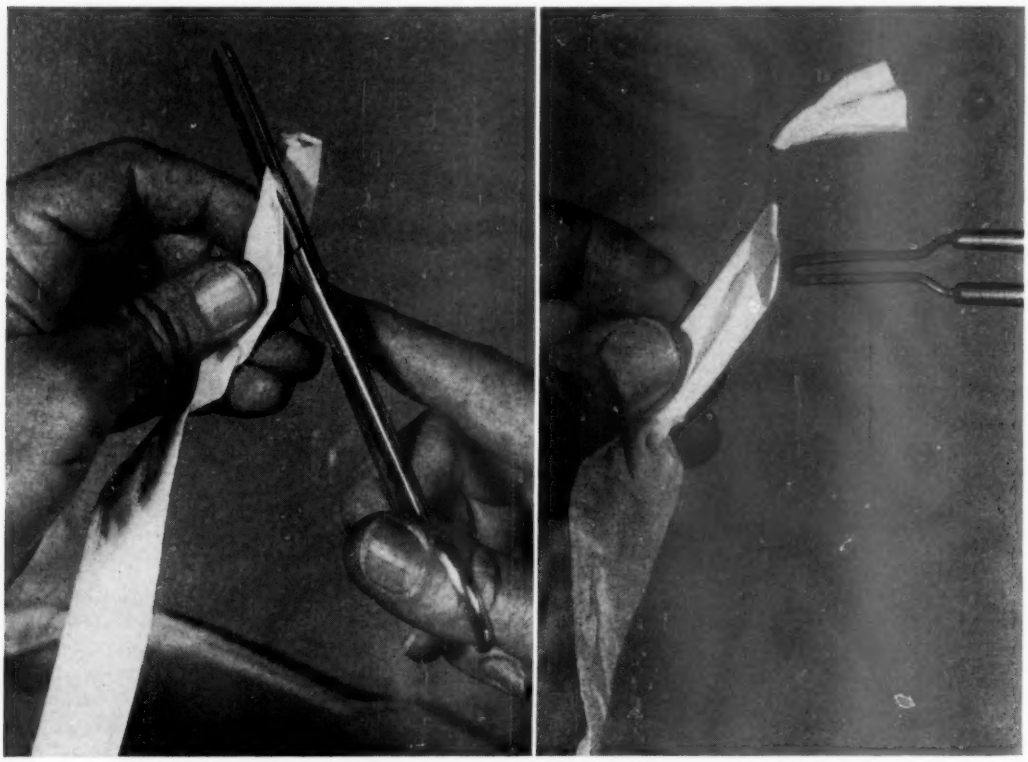
A.



B.

Fig. 4.—*A*, Photomicrograph of the full thickness of graft showing surface lining infiltrating through graft and the fibrous tissue envelope. *B*, High power field of same showing infiltration of fabric by fibroblasts.

bifurcation to the midpopliteal space. Replacement of the aortic bifurcation was accomplished using five different patterns of bifurcation grafts. Selection of the bifurcation in each case was on the basis of measurement at operation. The presently available five sizes permitted anastomoses without significant discrepancy in each case. Anastomoses were made by end-to-end suture utilizing a cuffed end of the tube or a suitably heat-fused edge. A continuous over-and-over suture technique was employed using 4-0 or 5-0 braided silk.



A. *B.*

Fig. 5.—*A*, Photograph of beveled cut being made in straight tube for use as by-pass. *B*, Heat fusion of graft end showing character of bevel in relation to the selvedges of tube.

In order to implant the ends of the straight tubes clinically as end-to-side by-passes, it was necessary to bevel them and fuse the resulting edges. The heat source used for fusion was an actual cautery loop carried into the operative field. The tube was cut on an appropriate angle with the seams arranged so that they would lie parallel to the fascial planes after implantation (Fig. 5, *A*). The cautery heated to a dull red was then passed approximately 1 mm. away from the Dacron, along the cut edge in order to fuse it without changing the contour of the cut (Fig. 5, *B*). After making the first anastomosis, the graft was passed through a subfascial or subcutaneous channel and cut off to length by direct approximation to the second anastomosis site. The length was gauged to provide for distinct tension on the graft. Heat fusion of this end may be easily accomplished with the wet material.

At the Research and Educational Hospital of the University of Illinois and St. Luke's Hospital, 15 femoral by-passes were done with the beveled

straight Dacron tube. The length of these varied from 20 to 36 cm., 10 being in excess of 30 cm. Four long by-passes were made by the New York group. Of these, two were in excess of 30 cm. In this group is represented a maximum degree of diffuse vascular change compatible with any attempt at arterial reconstruction. In 3 of the 19 cases, ischemic necrosis was present in toes or foot. Fundamentally, the use of Dacron tube in these patients was on the basis of not having a suitable artery or vein graft. The results of this clinical experience are tabulated in Table III, *A*. Seven of the grafts thrombosed immediately. Three were acutely thrombosed 4 days to 4 months after implantation, and two became occluded during the course of intractable wound infection aggravated by the foreign body. Seven remain patent and functioning for periods up to 7 months.

Four femoral by-passes with curved-end Dacron tubes, 15 and 20 cm. in length, were carried out in the Presbyterian Hospital. These 4 implants remained patent and functioning for periods of from 2 to 8 months (Table III, *B*).

TABLE III. CLINICAL RESULTS

A. FEMORAL BY-PASSES WITH BEVELED TUBE		
	ST. LUKE'S HOSPITAL AND UNIVERSITY OF ILLINOIS	PRESBYTERIAN HOSPITAL, N. Y.
Number	15	4
Length	20 to 36 cm.	20 to 35 cm.
Immediate thrombosis	3	4
Infected grafts	2	0
Late thrombosis	3	0
Number patent (2-7 mo.)	7	0

B. FEMORAL BY-PASSES WITH CURVED-END TUBE	
	PRESBYTERIAN HOSPITAL, N. Y.
Number	4
Length—15 cm.	2
20 cm.	2
Patent (2-8 mo.)	4

C. AORTIC BIFURCATION GRAFTS		
	ST. LUKE'S HOSPITAL AND UNIVERSITY OF ILLINOIS	PRESBYTERIAN HOSPITAL, N. Y.
Aneurysm	11	3
Ruptured aneurysm	2	2
Leriche syndrome	1	0
Mortality	3	2
Total 19 grafts All grafts patent 2 to 9 months 5 autopsies showed patent grafts		

A total of 19 implantations have been done, 14 at the University of Illinois and St. Luke's Hospital, and 5 at Presbyterian Hospital. Eighteen were for aneurysm of the abdominal aorta, 4 of which were acutely ruptured. One was implanted in a case of chronic thrombosis of the aortic bifurcation. Five deaths, not related to the graft, occured in aneurysms. Of these, 3 were in patients with ruptured aneurysms, and 1 died in 9 days with massive myocardial infarct. In each instance, an autopsy revealed patency of the graft. The 14 remaining patients have been observed for periods up to 9 months and show clinical evidence of complete patency of the grafts (Table III, C).

DISCUSSION

The original purpose of this project was to produce seamless, prefabricated, aortic bifurcations in various sizes for the convenience, strength, and availability which would result. Selection of Dacron as the material in preference to nylon was on the experimental basis that nylon loses tensile strength after implantation for a year or more.[4-5] The strength of the selvedge type of seam and the fact that its porosity is the same as that of the adjacent fabric were predictable advantages of this method. The development of straight tubes was initially for the purpose of testing this construction in animals. Early clinical use of Dacron tubes was justifiable on the basis that it was confined to long replacements for which homologous arteries or autogenous veins were not available.

The rate of success in by-passes using the straight tubes in man has been understandably low, there being 12 immediate and late failures in 19 cases. On the basis of the extensiveness of the disease, which necessitated these long tubes, and the presence of ischemia in the limbs of these patients which indicates diffuse disease, we may raise the question if this percentage would be improved upon through the use of any type of graft. A superiority of the curved-end tubes as by-passes is suggested by the four successful cases with no failures. The length of the tubes, however, were limited to 15 and 20 cm. and were consequently implanted in patients with less extensive disease. The future use of this type of tube in longer lengths will determine if they are actually superior.

The uniform functional success of the 19 fabric bifurcations is not unexpected in view of the results of others who have used Vinyon-N, Dacron, and Orlon fabric. Implantation of these grafts presented no technical difficulties when either the cuff or the fused end was used for anastomosis. A variable amount of bleeding occurred with some of the bifurcations, when blood flow was first re-established. This was minimal in most of the cases, particularly if the graft was allowed to become blood-soaked and then stripped free of clots before implantation. Considerable variation in blood loss through the fabric was observed despite uniform preparation as has been the case with prostheses of other fabrics.

A 15 per cent incidence of thrombosis of experimental grafts placed in the abdominal aorta of dogs was observed, while placement in the thoracic aorta was uniformly successful. This may be due to occasional wrinkling because of insufficient tension of contracture of hematoma or to the lesser caliber of the abdominal aorta. An even lower success rate has been reported with replacements of various types in the carotid and femoral arteries of dogs.

On the basis of our combined experience, we believe that this construction of prosthesis is sound; that the functional success rate is still largely influenced by the type of case in which it is used; and that technologic improvements are to be expected. In the latter regard, experiments are in progress designed to reduce amount of blood loss through the fabric without sacrificing porosity.

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Discussion

DR. W. STERLING EDWARDS (Birmingham, Ala.).—I certainly want to compliment Drs. Julian and Deterling on this nice piece of work. I certainly agree that efforts should be made toward the development of prefabricated grafts rather than individually tailored grafts. I got the impression yesterday in discussions on Ivalon that some of the difficulties in the variability of different opinions concerning its safety and effectiveness was due to the fact that each individual was preparing his own tubes in the operating room. Therefore, it seems likely that prefabrication of a well-tested graft may have definite advantages.

We have also worked upon a prefabricated bifurcation, and we have tried to incorporate one additional factor that may be of some help. That is the factor of noncollapsibility of the bifurcation tubes similar to the noncollapsibility and nonkinking property in the long straight tubes which we have previously reported. (Slide.) This is a bifurcation tube of braided nylon with crimped limbs and a straight aortic segment. This is a relatively porous tube despite silicone treatment and we always preclot it in a special way which is very helpful. We remove 50 c.c. of blood from the patient's aorta with a No. 22 needle, place it in a glass beaker, and soak the outside of this graft with blood. The silicone coating of the tube does not allow the blood to enter the lumen of the tube, and therefore, there is no danger of internal thrombosis. This preclotting seals the pores completely so that after implantation there is almost no blood loss at all coming through the walls of the tube itself. This tube was constructed with crimped limbs so that a bifurcation by-pass technique could be used in patients in the following manner.

(Slide.) This is actually the picture drawn from the first case in which this was used and a by-pass constructed from the aorta just below the renals down to the superficial femoral vessels with good return of pulses in the feet.

(Slide.) This shows the actual graft after the implantation in this patient. The two incisions in the upper thighs are shown on the right and the anastomoses to the abdominal aorta on the left. We have carried out three such by-passes with success and have used this graft to replace an aneurysm one time.

DR. ARTHUR H. BLAKEMORE (New York, N. Y.).—I want to compliment the group for eliminating the seam, at least as far as the inside of the tube is concerned. I think that it is a very important achievement.

I wish to report on the efforts we have been making along the line of braided tubes. What we wait for now is delivery of a production machine for the "Y" tube, which is being held up, I understand, because of basic materials that they need to make the machine. It is hoped that it will come through because the experimental machine for "Y" tubes seemed to be producing a satisfactory bifurcation. We have a production machine for the straight tubes, but, of course, the "Y" tubes are in great demand. There is one feature among others that would seem, at least in the smaller diameter tubes, to be of great advantage for the braid and that is in experimental work there has been no tendency toward wrinkling of the braided tube when imbedded, after long periods. These tubes are made of braids that apparently have an elasticity or give in different directions, which perhaps prevents wrinkling.

I think that we are all making progress—very necessary progress.

DR. HARRIS B. SHUMACKER, JR. (Indianapolis, Ind.).—I would like to recount an interesting experience which demonstrates the fact that one needs not only the new fibrous blood vessel which is laid down about the plastic prosthesis, or the homograft, but also needs preservation of the original plastic fabric or the elastic layer of the homograft if dilatation is to be avoided.

Several years ago I removed an aneurysm from a man and replaced the segment with a plastic graft. In this case the iliac extensions were very short and the aortic limb was quite long. The patient did very well. Almost 2 years after implantation, however, he suddenly had an occlusion of circulation to the right lower extremity. One could feel the right iliac pulse in this thin man very well, but no pulses could be felt distally. Obviously, the occlusion was near the distal end of the common iliac artery. He was most reluctant to be operated upon, but I was convinced that he would otherwise lose his limb. I finally succeeded in getting permission to explore him only by telling him that I thought the operation would be much simpler than the previous one and would likely necessitate only the removal of the thrombotic mass from within the artery, or the insertion of a short graft.

Upon exploration, the new aorta and the new short iliac limbs surrounding the original graft were not abnormal in any way. It looked like a perfectly normal aorta with its bifurcation and no abnormality of size or consistency or even of pulsation was noted. However, he had developed definite dilatation of the common iliac artery on the left side beyond the site of the graft. On the right side there was only slight dilatation of the common iliac artery and it was obstructed at its distal end. I opened the right iliac artery below the grafted area and there a fresh thrombus superimposed upon a heavy calcific deposit near the bifurcation was found to be completely obstructing the lumen. In removing the obstructing mass, I carried out a more extensive thromboendarterectomy than I had planned to do. In dissecting the thickened lining membrane upward, I inadvertently separated the plastic limb of the original graft from the outer fibrous coat. I did not know what to do about it, and with further manipulation I was soon convinced that I could not tack it back securely in any way. Of course, I would have excised his aorta and iliac arteries and put in a new graft had I not promised that I would not do an extensive procedure. Under the circumstances, I had to do the next best thing, which turned out to be a good experimental procedure. I loosened the entire plastic graft from the new aorta and both iliacs much as one would do in carrying out a thromboendarterectomy, and I pulled the entire plastic lining out through the right common iliac arteriotomy. At the completion of this procedure things looked good. There was excellent circulation to the right lower extremity. Naturally, I was very much concerned. I made the patient promise, since he didn't want to be reoperated upon, that I could see him frequently. He was thin enough that I could palpate the aortic bifurcation well, and I measured the width of the pulsation at the bifurcation at regular frequent intervals. Then I had to be out of the city. About three and a half months after his operation, he was admitted to the hospital with a sudden onset of abdominal pain. He obviously now had a fairly large aneurysm. I was able to re-explore him. Whereas for nearly 2 years after his original operation he had maintained a perfectly normal-appearing aorta and bifurcation with the plastic lining inside the new fibrous blood vessels, he had now developed within a very short period of time a diffuse aneurysmal dilatation of the aorta and the common iliac arteries. After excising the aneurysm, a new plastic graft was inserted and he is quite well six months later. I suspect that we shall have to rely on the persistence of the plastic fabric to give strength to the vessel and obviate aneurysmal dilatation, and I also expect that the preservation of the elastic layer of the homograft will similarly be necessary in order to prevent untoward results.

DR. PAUL W. SANGER (Charlotte, N. C.).—I have a similar product to sell only it is a different model. We are using seamless knit grafts which the Research Department of the Textile School of North Carolina State College fabricates for us. In the experimental animal we have not seen much difference between the orlon and nylon grafts. There is some theoretical objection to nylon because it has been shown that nylon absorbs more water than the other synthetics. This creates the possibility that it may later stretch. We have not noted this in the experimental animal. It must be remembered that, as of this date, the Federal Pure Food and Drug Department recognizes only nylon as being acceptable for human use. These synthetics are used only in individuals who otherwise could not be saved; therefore I feel we are making an unnecessary issue. After all, these patients eventually die of the generalized disease and not of the lesion per se for which they were operated upon, provided of course they recover from the original surgery.

The observation commented upon this morning about some individuals bleeding more than others has been our experience also, and I can well understand how the same material may react differently in the individual in Texas from the patient in Charlotte. By the same token, orlon may differ from nylon in different individuals. We have found that attempting to preclot the prosthesis is advantageous. This we do by dropping the graft into the abdominal cavity to let it absorb the blood and serum before anastomosing it. The advantage of the knitted material is the absence of a seam. It does not fray easily and hence requires no cuff. Most of our experience has been with the orlon tube. Recently we have turned our attention to grafts made of two strands—one of orlon and the other of nylon (150 denier each).

After inserting a prosthetic graft the distal clamp is released and the integrity of the suture line determined. The graft is covered with dry sponges and the proximal clamp slowly released. Frequently it is necessary to reapply the clamp many times before annoying seepage actually stops. In a minute or two the bleeding completely ceases.

DR. RALPH A. DETERLING, JR. (closing).—I wish to thank Drs. Edwards, Blakemore, Shumacker, and Sanger for their comments. They have emphasized some important points which merit attention.

With regard to Dr. Edwards' remarks on prefabrication, I believe there is reasonable value in having readily available a stock of prepared replacements—bifurcations and curved or straight tubes. With a sufficient variety of sizes, most needs can be met. A seamless prosthesis or one with woven external seam, such as that which we have described, has the advantage of strength greater than that in grafts with seams made with the ordinary sewing machine. Occasionally a sewn seam has ruptured after implantation. Nevertheless, in patients requiring a prosthesis of an unusual size or shape or in institutions with rare need for a replacement, flat fabric will continue to be of value.

Dr. Blakemore has voiced approval of seamless synthetic replacements. While our prosthesis has no internal seams, I am not certain that seams do contribute significantly to thrombosis. We have implanted grafts with six vertical seams into the thoracic aorta of dogs without thrombosis developing. I believe that other factors are more important in the maintenance of functional success.

We have noted little or no wrinkling of fabric grafts at time of autopsy some months following implantation in animals. Wrinkling seemingly contributes to failure, especially if blood flow is slow or at low pressure. If grafts are implanted under moderate tension (before re-establishing blood flow) and if all oozing of blood from the suture lines and fabric has been controlled before closing the wound, wrinkling should not be a problem.

(Slide.) This is the appearance of a by-pass graft of dacron fabric at the time of operation. While the amount of blood loss through the fabric may vary with the individual patient, there may also be variability in porosity of a fabric in a single yard. Porosity of a fabric may be an important factor in assuring functional success of a fabric prosthesis. However, we think flexibility is perhaps more vital, since we have observed as good a functional result with fabric of very low porosity as with one having marked porosity. This question is not yet settled.

(Slide.) This is an interesting case. The arteriogram shows a functioning by-pass homograft. In this patient with segmental arteriosclerotic occlusion of the femoral artery, a fabric by-pass was implanted after injecting heparin by polythene catheter into the artery distally. At the time of injection a plaque was loosened below the site of the lower anastomosis and the artery thrombosed at this site postoperatively. Two weeks later an arteriogram revealed a patent by-pass but a 6 cm. occlusion distally. A homograft was used to bridge this segment and the patient now has a warm foot with pedal pulses. At the second operation, the fabric graft was exposed and resembled a normal pulsating artery grossly.

VASCULAR PROSTHESES

Report of the Committee for the Study of Vascular Prostheses of the Society for Vascular Surgery

Oscar Creech, Jr., M.D., Chairman, Ralph A. Deterling, Jr., M.D., Sterling Edwards, M.D., Ormand C. Julian, M.D., Robert R. Linton, M.D., and Harris Shumacker, Jr., M.D.

WITHIN the past 5 years increasing indications for excision or by-pass procedures in the treatment of aortic and peripheral arterial lesions have created a demand for vascular substitutes out of proportion to the available supply of arterial homografts. Thus, the need for a suitable vascular prosthesis made of commercially available materials has become acute. As a result of this need a variety of materials, largely synthetic, has been investigated in experimental animals and in humans. From this experience, significant data relating to operative technique and the early behavior of these materials after transplantation have been derived. In spite of these studies, however, there has been considerable difficulty in evaluating the results obtained with synthetic vascular substitutes, largely because of the many different types of material in use and the fact that no one investigator's experience is large enough to be conclusive. Recognizing the importance of this problem, the Society for Vascular Surgery at its annual meeting in 1955 appointed a Committee for the Study of Vascular Prostheses. This report is concerned with the findings of this committee.

The study was conducted along the following lines: (1) Available data were collected relating to types of materials used, including the composition and properties of the crude materials, methods of fabrication, and properties of the finished prostheses. (2) The results of experimental and clinical trials with these materials were collected. (3) The failures were analyzed in terms of material, technical features and other factors contributing to failure. (4) Observations on the biologic fate of synthetic prostheses after transplantation were assembled. (5) The opinion of members of the Society and others in vascular surgery was determined with regard to the essential features of synthetic vascular substitutes.

To commence this study, a questionnaire was sent to each member of the Society for Vascular Surgery and others known to have an interest in this field. As anticipated, the response to the questionnaire was varied and not overwhelming. Thus, of 127 questionnaires sent out, 67 were returned. There were 29 replies which indicated that the surgeon questioned did not use synthetic prostheses, and 12 were of no value because of the way in which the questionnaire was completed. Satisfactory data were received from 27 surgeons and this material comprises the statistical portion of the report.

Read at the tenth annual meeting of the Society for Vascular Surgery, Chicago, Ill., June 10, 1956.

RESULTS OF QUESTIONNAIRE

Materials.—There were 8 basic materials used for vascular substitutes, as follows: Vinyon-N, nylon, Orlon, Dacron, Ivalon, Teflon, Fortisan, and stainless steel. These materials were fabricated into 17 types of prostheses, 2 of which were nonporous and the remainder were porous. In 3 instances the prostheses were made commercially, while the rest were constructed by the surgeon. A description of the essential features of these materials follows.

A. CHEMICAL PROPERTIES.—

1. *Vinyon-N.*—Vinyon-N was introduced about 1950 and was the material selected for the first successful fabric graft. The polymer is 60 per cent vinyl chloride and 40 per cent acrylonitrile. The yarn in multifilamentous, each filament measuring 16 to 18 micra and has an irregular shape on cross section.

2. *Nylon*—This substance was produced in 1938 and is a straight-chain condensation product of an amino group with a carboxyl group with loss of a molecule of water. The fabric is generally constructed of multifilament yarn. The filament has a diameter of 16 to 43 micra and is round on cross section.

3. *Orlon.*—Orlon became available about 1948 and is an addition type of polymer made possible through the use of polyacrylonitrile. The yarn is generally multifilamentous with the filament having a clover-leaf or dogbone shape and a diameter of 16 micra.

4. *Dacron.*—This was introduced into America in 1946 although it had been developed in 1939 as a polyester polymer of ethylene glycol and terephthalic acid. The yarn is multifilamentous, each filament measuring 7 to 28 micra and being round in shape.

5. *Teflon.*—This has been available as multifilament yarn since 1954 but is a much heavier denier than that of the previously described materials. It has a "slippery feel" and because of inert carbonaceous impurities it has a dark brown color. The filaments are round on cross section.

6. *Ivalon.*—This substance is a vinyl chloride polymer produced in sponge form about 1952. As such, the material is stiff when dry but very resilient when wet. The material is easily compressed and maintains its form after being subjected to heat.

7. *Fortisan.*—This is a regenerative cellulose product and has limited experimental trial as a multifilament yarn.

8. *Stainless Steel.*—Stainless steel, a high temperature alloy, has been available for fabrication in fine caliber, multifilament strands.

B. PHYSICAL CHARACTERISTICS.—

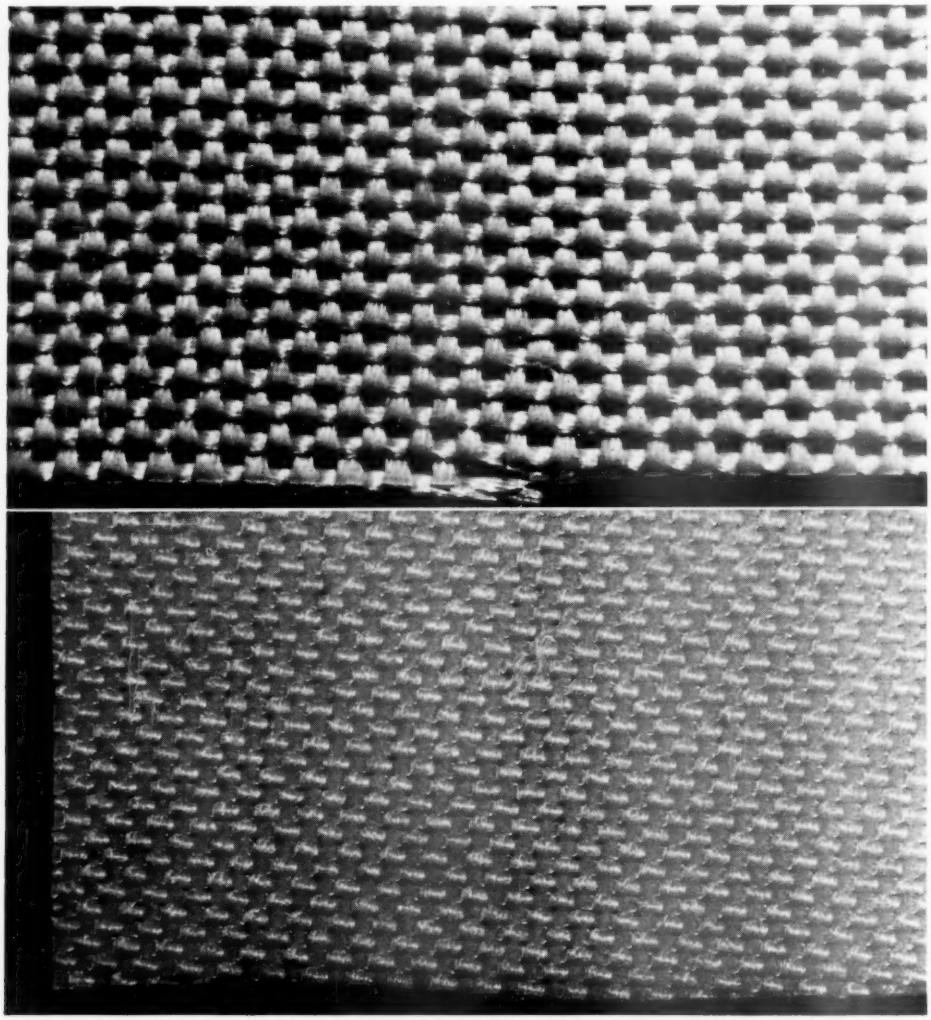
1. *Specific Gravity.*—Among the synthetic materials tried, Teflon is the heaviest with a specific gravity of over 2.1. Most of the other materials range between 1.14 and 1.38.

2. *Moisture Regain and Tensile Strength.*—The degree of water absorption is now believed to be of importance in influencing behavior of synthetic materials in mammalian tissues. Evidence has accumulated from experimental trial of these materials for joint replacements as well as for blood vessel prostheses which suggests that reduction in tensile strength and in stability of the polymer with time may be related to water absorption. In line with this theory there has been observed a significant reduction in tensile strength of nylon yarn implanted in mammalian tissues for over one year. The moisture regain in nylon is up to 4.5 per cent. On the other hand, far less loss of tensile strength has been observed with Vinyon-N, Dacron, or Teflon, in which water absorption is less than 0.4 per cent. Water absorption in Ivalon is minimal, as is that of stainless steel, but potential sources of weakness of these materials arise from other causes. Whereas, synthetic yarn is continuous and has predictable strength related to its denier, Ivalon is amorphous and its integrity is broken by air spaces. Stainless steel has the tendency of most stiff metals to "fatigue" at sites of continuous binding stress. The wet strength of all these materials is 85 to 100 per cent, being lowest in nylon.

3. *Elasticity.*—Although a minimal elasticity is present in synthetic yarn, the gross characteristics of elasticity and flexibility are more a function of construction. Ivalon exhibits significant elasticity in contrast to stainless steel strands.

4. *Electrostatic Charge.*—It has been proposed that surface electric charge of a synthetic material might influence the clotting mechanism through the surface attraction of platelets. Among the synthetic yarns, nylon exhibits the greatest voltage, whereas Vinyon-N has considerably less. The values for Dacron and Teflon have not been available. Actual experience would discount this factor as being vital, however.

A.



B.

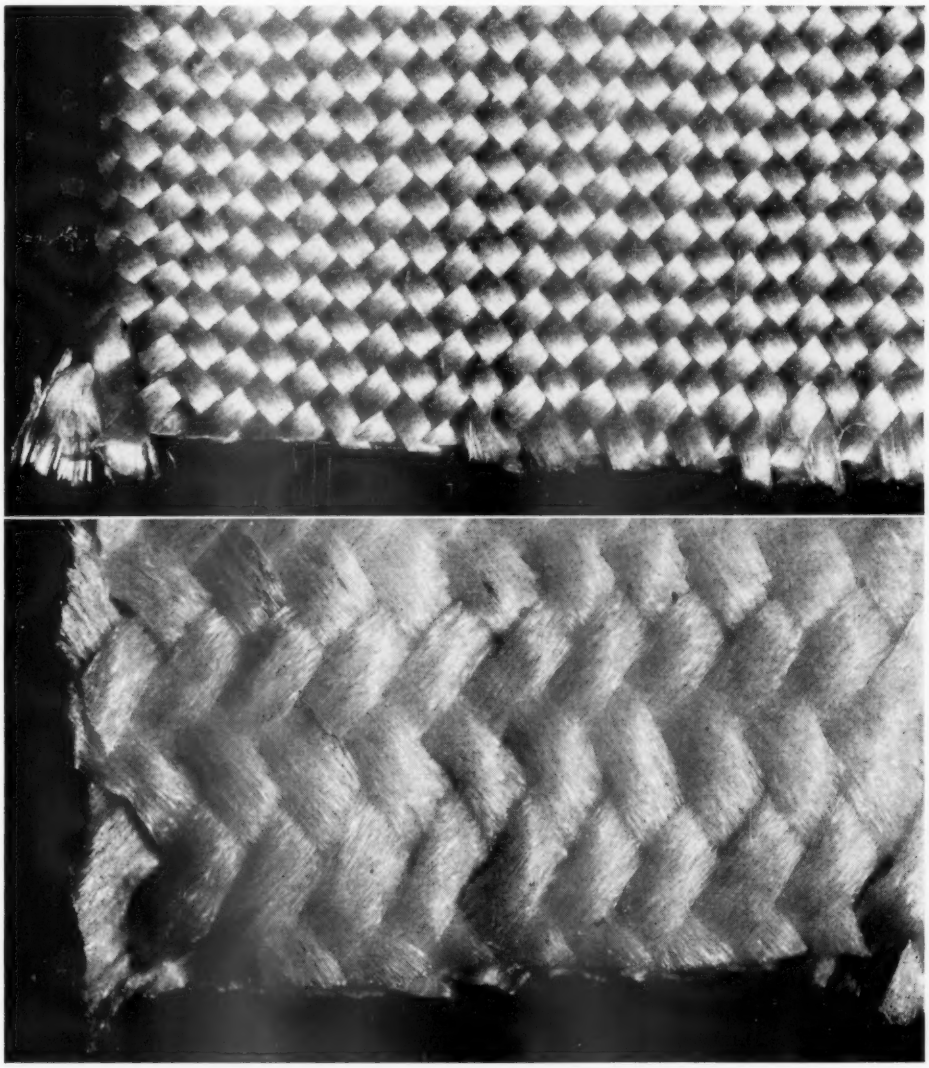
Fig. 1.—*A*, Photograph of nylon fabric of taffeta weave construction (Shumacker). *B*, Dacron fabric, taffeta weave, which has been calendered (Deterling). The yarn is flattened and the interstices are almost completely closed by the calendering process.

5. *Reactivity in Tissues and Carcinogenic Properties.*—Scales have suggested that criteria for suitability of synthetic materials in mammalian tissues should be: (1) no physical modification by exposure to tissue fluids, (2) chemical inertness, (3) inertness in respect to foreign body effect, (4) noncarcinogenic, (5) nonallergenic, (6) stability under continuous mechanical stress, (7) ease and low cost of fabrication, and (8) ease of adequate sterilization. In regard to synthetic materials there is evidence that minimal response is elicited by Vinyon-N, Dacron, and Teflon, whereas moderate reaction is observed with Orlon, nylon,

and Ivalon. The irritation to Fortisan is not yet completely evaluated. Low-grade reaction may be expected to stainless steel. There is no good evidence that properly cleansed synthetic materials among those described in this report produce an allergic state.

Of great medical interest and medicolegal importance is the question of carcinogenesis. Recent reports by investigators studying this aspect in rodents indicate positively that synthetic fabrics of nylon, Vinyon-N, and Dacron have never produced a sarcoma. Testing has not been completed with fabrics of Orlon or Teflon. By contrast, films of these materials are capable of developing sarcoma in rats and mice, but so are many other films as well as cover slips of glass. Stout has observed that malignancy has never been reported in his knowledge as a result of synthetic films or fabric being implanted into a human being.

A.



B.

Fig. 2.—*A*, Photograph of a portion of an Orlon prosthesis of braid construction (Blakemore). *B*, A braided nylon prosthesis which has been treated with formic acid (Edwards).

6. *Sterilization.*—Among the yarns discussed here, Vinyon-N was found to shrink and stiffen when subjected to a standard technique of autoclaving. Changes in nylon, Dacron, Orlon, Ivalon and Teflon were negligible. Results with fortisan are not recorded. Stainless steel is stable to autoclaving. Cold sterilization by antiseptic solutions did not affect any of these materials.

C. CONSTRUCTION.—

1. *Weaves.*—

a. *Flat fabric:* The taffeta weave has been most often employed for blood vessel replacement. The regular over-and-under weave results in a smooth surface and even less texture can be achieved by a calendering process (Fig. 1, *A* and *B*). It is of interest, however, that experimental results show functional success to be about equal with all taffeta fabrics regardless of calendering. There is obviously considerable latitude permitted in surface texture in so far as thrombosis is concerned, provided a streamline blood flow of sufficient volume and pressure is present.

b. *Prefabricated prostheses:* Seamless tubes of a taffeta weave are now available in nylon, Dacron, or Teflon. Prefabricated tubes or bifurcations in all desirable sizes and shapes are also available in Dacron taffeta. The latter prostheses are produced by duplication weaving. Their selvage seams are not discernible from the inner surface and have the same strength as the fabric. The advantage of the seamless or duplication type of prosthesis is ready availability and greater intrinsic strength. Because of the lack of "body" and shape-retaining properties these prostheses may wrinkle or buckle after implantation and this feature may constitute a distinct disadvantage to the use of bifurcation prostheses made of this material.

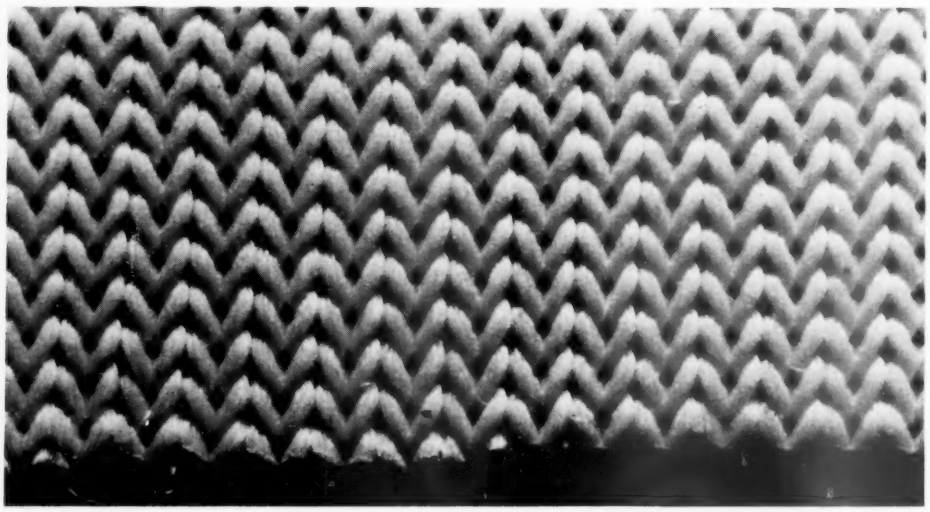


Fig. 3.—Photograph of a knitted Orlon prosthesis (Sanger).

2. *Braid.*—This construction has been employed less commonly because of the usually thick walls and rough surface. However, functional success of a high degree has been achieved with examples of this type (Fig. 2, *A*). Braided tubes of heavier denier yarn have been crimped to permit greater flexibility in cross flexion areas of the body (Fig. 2, *B*). The long-term results will indicate whether satisfactory functional results will be maintained despite greater thickness and rougher surface than the taffeta weaves.

3. *Knit.*—Knitted prostheses, usually of Orlon yarn, are available. The surface is generally smoother than that achieved with braiding. However, since each thread is looped upon itself, at which point there is a mechanical stress, the looped strength of synthetic yarn is less than in straight form (Fig. 3). Trial of a nylon tulle construction revealed breakage of the threads at the looping after continued use, and whether this danger exists with a regular knit is not predictable at the present time.

4. *Ivalon Sponge.*—This material is of an amorphous character perforated by air spaces. The original advantages were cited as nonabsorbability and porosity permitting ingrowth of fibroblast through the air spaces. However, use of the noncompressed material revealed insufficient strength for use in arteries. With tight compression the wet material resembles the normal aorta in handling qualities but has little porosity left for infiltration by fibroblasts. It is not yet known whether the compressed material will have sufficient inherent

strength to resist arterial pressure for long periods of time. A favorable characteristic is the molding quality. Ivalon may be compressed on a form of desired shape and size and will retain this contour after treatment in boiling water or by autoclaving.

5. *Mesh.*—There has been limited experience with stainless steel mesh but the great porosity as compared with other constructions already described as well as its rigidity would appear to constitute distinct disadvantages. The open framework may permit significant blood loss at the time blood flow is established through the graft (Fig. 4). This is a disadvantage to any porous type of replacement regardless of construction. Such loss is combated by: (1) pre-clotting, (2) intermittent re-establishment of blood flow, or (3) treatment of material with plastic or organic film to reduce porosity.

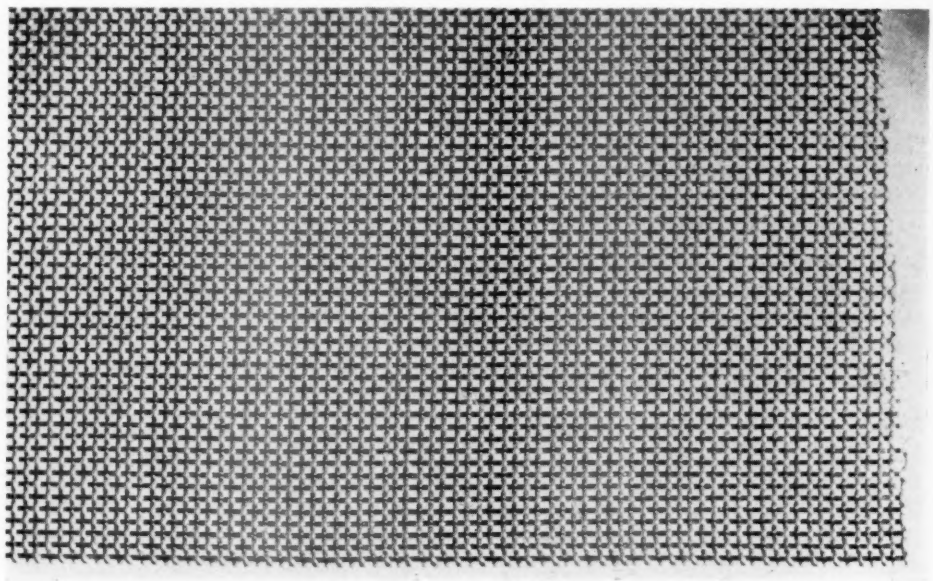


Fig. 4.—Photograph of stainless steel mesh (200) (de Takats).

D. Results of Use.—

1. *Vinyon-N.*—In experimental studies this material was used to replace segments of the aorta in 170 instances and peripheral vessels in 23 (Table I). Of the experiments involving the aorta, 146 (86 per cent) were successful and 24 (14 per cent) failed, while of the prostheses used in peripheral vessels, 14 (61 per cent) were unsuccessful.

Table I. Vinyon-N

SITE	TYPE	NUMBER	SUCCESSFUL	UNSUCCESSFUL
Aorta	Experimental	170	146 (86%)	24
	Clinical	49	45 (92%)	4
Peripheral arteries	Experimental	23	9 (39%)	14
	Clinical	15	10 (67%)	5

Clinically, Vinyon-N prostheses were implanted into the aorta in 49 cases, 45 (92 per cent) of which were successful (Table I). Among the 4 failures, 2 were due to disruption of the prosthesis about 30 months after implantation and 2 were due to thrombosis. In peripheral arteries 15 prostheses of Vinyon-N were employed clinically, 10 (67 per cent) of which were successful. All of the failures were due to thrombosis.

2. *Nylon.*—Nylon yarn was used for the construction of 5 types of prostheses. *Nylon fabric* was employed in 72 experiments in which segments of the aorta were replaced, with successful results in 60 (83 per cent) (Table II). This material was used in only 4 experiments involving peripheral arteries, 2 of which were successful.

TABLE II. NYLON FABRIC

SITE	TYPE	NUMBER	SUCCESSFUL	UNSUCCESSFUL
Aorta	Experimental	72	60 (83%)	12
	Clinical	16	14 (87%)	2
Peripheral arteries	Experimental	4	2 (50%)	2
	Clinical	2	1 (50%)	1

Clinically, nylon fabric prostheses were implanted in the aorta in 16 cases (Table II). There were 2 (13 per cent) failures in this group, one from disruption of the prosthesis at the seams at 15 months and one from the development of a fistula between an iliac anastomosis and the ileum. This material was used in peripheral arteries in only 2 cases, one of which was successful. The failure was due to thrombosis of the graft.

TABLE III. NYLON-POLYETHYLENE

SITE	TYPE	NUMBER	SUCCESSFUL	UNSUCCESSFUL
Aorta	Experimental	31	23 (74%)	8
	Clinical	11	10 (91%)	1

Nylon fabric rendered impervious by the application of a polyethylene film was used in 28 experiments involving resection of the aorta, 23 of which were successful (Table III). Clinically, this material was used to replace segments of the aorta in 11 cases, 10 of which were successful. In the one case classified as a failure, infection developed about the prosthesis.

TABLE IV. NYLON FILTER FABRIC

SITE	TYPE	NUMBER	SUCCESSFUL	UNSUCCESSFUL
Aorta	Experimental	32	31 (97%)	1
	Clinical	21	21 (100%)	0

Nylon filter fabric was utilized in 32 animal experiments to replace excised segments of the aorta (Table IV). All but one of these were successful. This material was employed in 21 clinical cases for aortic replacement, all of which were successful.

TABLE V. BRAIDED NYLON–CRIMPED

SITE	TYPE	NUMBER	SUCCESSFUL	UNSUCCESSFUL
Aorta	Experimental	75	74 (99%)	1
	Clinical	4	4 (100%)	0
Peripheral arteries	Experimental	–	–	–
	Clinical	70	56 (80%)	14

Braided nylon tubes (crimped) were employed in 75 experiments involving the aorta, only one (2 per cent) of which was reported as a failure (Table V). This number does not include the instances of thrombosis occurring in the early period after operation since it was felt that these failures were not failures of the prostheses but were probably a result of technical factors. Clinically, braided nylon tubes have been used in 4 cases to replace excised segments of the aorta and in each instance were satisfactory. This prosthesis was utilized in peripheral arteries in 70 clinical cases, and in 56 (80 per cent) the results were reported to be successful at intervals varying from 1 week to 18 months after operation. There were 14 (20 per cent) failures in this group.

TABLE VI. TOTAL RESULTS WITH NYLON PROSTHESES

SITE	TYPE	NUMBER	SUCCESSFUL	UNSUCCESSFUL
Aorta	Experimental	210	188 (89%)	22
	Clinical	52	49 (94%)	3
Peripheral arteries	Experimental	4	2 (50%)	2
	Clinical	72	57 (79%)	15

3. *Dacron.*—

Dacron taffeta prostheses were used in 112 experiments involving the aorta (Table VII). Failure occurred in 8 (7 per cent) and in each instance was due to thrombosis. This material was used in peripheral arteries in only 4 experiments, 2 of which were successful. The failures were also due to thrombosis. Prostheses made of Dacron taffeta have been employed in 32 clinical cases to replace segments of the aorta (Table VII). One failure (3 per cent) occurred and was due to thrombosis of the common iliac branch of a bifurcation prosthesis. In peripheral arteries, Dacron taffeta was used experimentally in 4 instances, 2 of which were successful. Clinically, 22 (56 per cent) of 39 cases were successful. In each instance, failure was due to thrombosis.

TABLE VII. DACRON TAFFETA

SITE	TYPE	NUMBER	SUCCESSFUL	UNSUCCESSFUL
Aorta	Experimental	112	104 (93%)	8
	Clinical	32	31 (97%)	1
Peripheral arteries	Experimental	4	2 (50%)	2
	Clinical	39	22 (56%)	17

4. *Orlon.*—

Orlon fabric (taffeta) coated with vinyl plastic was used as a vascular prosthesis in 12 clinical cases (Table VIII). One of these patients (8 per cent) died from hemorrhage 8 months after operation as a result of erosion of the proximal end of the prosthesis into the duodenum. A second patient died almost 2 years after operation from a coronary occlusion, and this prosthesis was examined grossly and microscopically and the results appear later in the report.

TABLE VIII. ORLON TAFFETA COATED WITH VINYL PLASTIC

SITE	TYPE	NUMBER	SUCCESSFUL	UNSUCCESSFUL
Aorta	Experimental	–	–	–
	Clinical	12	11 (92%)	1

TABLE IX. ORLON TAFFETA

SITE	TYPE	NUMBER	SUCCESSFUL	UNSUCCESSFUL
Aorta	Experimental	37	28 (76%)	9
	Clinical	15	14 (93%)	1
Peripheral arteries	Experimental	–	–	–
	Clinical	8	5 (63%)	3

Prostheses made of *Orlon taffeta* were used in 37 experiments to replace segments of the aorta (Table IX). Nine of these experiments were unsuccessful. Seven of the failures were due to thrombosis and 2 were a result of disruption of the prosthesis. This material was used to replace segments of the aorta in 15 clinical cases, one (7 per cent) of which was unsuccessful due to hemorrhage (Table IX). Of 8 instances in which Orlon taffeta was used to replace peripheral arteries, 5 (63 per cent) were successful, and 3 were unsuccessful. Each of the failures was a result of thrombosis of the graft, in one of which infection was superimposed resulting in sequestration of the prosthesis.

TABLE X. ORLON KNIT

SITE	TYPE	NUMBER	SUCCESSFUL	UNSUCCESSFUL
Aorta	Experimental	30	30 (100%)	0
	Clinical	72	70 (97%)	2
Peripheral arteries	Experimental	–	–	–
	Clinical	11	1 (9%)	10

Knitted Orlon prostheses were employed as aortic replacements in 30 experiments (Table X). There were no failures in this group. This material was used in 72 clinical cases for aortic replacement, 70 (97 per cent) of which were successful (Table X). One patient died

at 8 months of other causes and the prosthesis was removed at necropsy and examined grossly and microscopically. The results of this study are presented elsewhere in the report. The 2 failures were due to hemorrhage. In peripheral arteries, prostheses made of knitted Orlon were used in 11 cases, 9 (91 per cent) of which were unsuccessful. In each instance failure was a result of thrombosis.

Braided Orlon Prostheses were used in 3 experiments to replace segments of peripheral arteries, but none of these were successful.

TABLE XI. TOTAL RESULTS WITH ORLON PROSTHESES

SITE	TYPE	NUMBER	SUCCESSFUL	UNSUCCESSFUL
Aorta	Experimental	67	58 (87%)	9
	Clinical	99	95 (96%)	4
Peripheral arteries	Experimental	3	0 (0%)	3
	Clinical	19	6 (32%)	13

5. *Fortisan.*—

Fortisan was utilized as an aortic prosthesis in 40 experiments, 16 (40 per cent) of which were unsuccessful (Table XII). In each instance the failure was due to occlusion of the prosthesis. This material was used successfully in one clinical case to replace a segment of aorta.

TABLE XII. FORTISAN

SITE	TYPE	NUMBER	SUCCESSFUL	UNSUCCESSFUL
Aorta	Experimental	40	24 (60%)	16
	Clinical	1	1 (100%)	0

6. *Ivalon.*—

Prostheses made of *polyvinyl sponge* were employed 41 times in experiments involving aortic replacement, 36 (88 per cent) of which were successful (Table XIII). This material was used experimentally in peripheral arteries in 30 instances, only 37 per cent of which were reported to be successful. Clinically, Ivalon aortic prostheses were used in 22 instances, 17 (77 per cent) of which were successful. This material was employed as a prosthesis for replacement of peripheral arteries in 14 instances, only 4 (28 per cent) of which were reported to be successful. The cause of failure was thrombosis and, in addition, 2 of these prostheses were infected.

TABLE XIII. IVALON

SITE	TYPE	NUMBER	SUCCESSFUL	UNSUCCESSFUL
Aorta	Experimental	41	36 (88%)	5
	Clinical	22	17 (77%)	5
Peripheral arteries	Experimental	30	11 (37%)	19
	Clinical	14	4 (28%)	10

7. *Teflon.*—

Teflon prostheses were used in 60 experiments to replace segments of the aorta, 5 (8.3 per cent) of which were unsuccessful (Table XIV). This material was utilized as an aortic prosthesis in one clinical case with excellent results. Similarly, it was used successfully as a replacement for peripheral arteries in one clinical case.

TABLE XIV. TEFLON

SITE	TYPE	NUMBER	SUCCESSFUL	UNSUCCESSFUL
Aorta	Experimental	60	55 (92%)	5
	Clinical	1	1 (100%)	0
Peripheral arteries	Experimental	–	–	–
	Clinical	1	1 (100%)	0

8. *Stainless Steel Mesh.*—

Stainless steel mesh tubes were used to replace segments of the aorta in 8 experiments, 5 of which were successful. The failures were all due to thrombosis.

In summary, 17 different types of vascular prostheses have been employed. Among 702 experiments in which prostheses were used as aortic replacement, 611 (87 per cent) were successful. In experiments involving peripheral arteries, 61 prostheses were used with failures in 83 per cent. Clinically, 256 synthetic aortic replacements were employed with success in 239 (93 per cent) (Table XV). Prostheses were used in peripheral arteries in 160 cases, 101 (63 per cent) of which were successful.

TABLE XV. TOTAL CLINICAL RESULTS

	AORTA	PERIPHERAL ARTERIES	TOTAL
Successful	239 (93%)	101 (63%)	333
Unsuccessful	17 (7%)	59 (37%)	76
	256	160	409

TABLE XVI. CLINICAL FAILURES WITH AORTIC PROSTHESES

1. Vinyon-N	Disruption	2
	Thrombosis	2
2. Nylon fabric	Disruption	1
	Iliac-ileal fistula	1
3. Nylon-polyethylene-nylon	Infection	1
4. Dacron taffeta	Thrombosis	1
5. Orlon taffeta	Hemorrhage	1
6. Orlon taffeta coated with vinyl plastic	Hemorrhage from erosion of duodenum	1
7. Orlon knit	Hemorrhage	2
8. Ivalon	Rupture	1
	Hemorrhage	1
	Thrombosis	1
	Aneurysm formation	2
	Total	17

ANALYSIS OF CLINICAL FAILURES

Aortic.—There were 17 failures among the 256 cases in which synthetic aortic replacements were used (Table XVI). There were 4 with Vinyon-N, 2 each from disruption and thrombosis. All of these failures occurred in the late follow-up period after the prostheses had functioned satisfactorily initially. There were 2 failures with prostheses made of nylon fabric, one due to disruption at the seams and the other due to the development of a fistula between the iliac portion of the prosthesis and the ileum. These complications also occurred late. There was one instance of infection in a case in which a prosthesis made of nylon reinforced with polyethylene was used. In this instance, death of the patient occurred 2½ months after operation as a result of rupture of a large aneurysm of the left common iliac artery. There was infection about the graft and disruption of the left iliac anastomosis could not be ruled out. The patient survived operation but succumbed to acute renal failure. At necropsy the prosthesis was found to be infected and this development may have been a factor in the death of the patient. There was one instance of thrombosis within an iliac branch of an aortic prosthesis made of Dacron taffeta. Fatal hemorrhage occurred in the early postoperative period following

implantation of an Orlon taffeta prosthesis. An Orlon fabric (taffeta) prosthesis rendered impervious by application of vinyl plastic to the external surface eroded the third portion of the duodenum and resulted in fatal hemorrhage in a patient 8 months after implantation. At necropsy the synthetic material was rigid, whereas at implantation it was soft and pliable. There were 2 deaths reported which were a result of hemorrhage from Orlon knit prostheses implanted into the abdominal aorta. In these cases, clotting in the interstices of the prosthesis failed to occur and fatal hemorrhage ensued. There were 5 instances of failure with Ivalon aortic prostheses. In one case, rupture occurred after operation had been completed and required removal of the Ivalon prosthesis and replacement with a homograft. In another case hemorrhage occurred

Fig. 5.—Photograph of an Ivalon aortic bifurcation prosthesis about one year after implantation. Both iliac portions of the prosthesis are acutely kinked causing complete obstruction of the lumen. There is almost no attachment of surrounding tissues to the prosthesis.

6 weeks after implantation and terminated fatally. There was one instance of thrombosis of the prosthesis. Thrombosis appeared to have resulted from kinking of the iliac limbs of the graft and became apparent clinically about 2 months after implantation. The prosthesis was removed one year later and the lumen was found to be completely obliterated (Fig. 5). It was observed in this case that the Ivalon was entirely unattached to the dense fibrous connective tissue surrounding it and microscopic examination failed to disclose any evidence of fibroblastic penetration. The formation of small aneurysms at the

junction of an Ivalon prosthesis with the iliac arteries was reported in 2 instances, and it was the opinion of the observer that the rigidity of the prosthesis had produced tension on the suture lines and was directly responsible for the development of the aneurysms.

Thus, out of 256 cases in which aortic prostheses were employed, there were 17 in which failure of function could be directly related to the prosthesis. Many other instances in which there were complicating factors associated with the use of an aortic prosthesis were reviewed and eliminated because there was not sufficient evidence to incriminate the prosthesis.

Peripheral Arteries.—Although the number of cases in which prostheses were employed successfully in peripheral arteries have been tabulated, an anlysis of the failures is not possible because of the incompleteness of the data which were received in most instances. For example, among the 14 failures reported with braided nylon tubes used to replace segments of peripheral arteries, in only 2 instances was the condition of the distal vascular bed at the time of operation stated. Thus, it was not possible to determine whether subsequent thrombosis of the prosthesis was related to the prosthesis itself or to lack of an adequate "run-off" area distal to the implant. Similarly, of 17 instances of failure of Dacron prostheses implanted into peripheral arteries, in only 3 were the precipitating causes of failure given. These were infection in 2 instances and kinking of the prosthesis with obstruction in one.

MORPHOLOGIC CHANGES IN SYNTHETIC VASCULAR PROSTHESES

Replies to the question regarding histologic appearance of the prostheses after implantation were essentially the same since, with 2 exceptions, all of the materials used were pervious. In general, it has been observed experimen tally that immediately upon establishing blood flow through the prosthesis, a layer of fibrin is deposited along the inner surface of the tube. At the end of one week there is a layer of granulation tissue surrounding the prosthesis and continuous with the adventitia of the recipient vessel. Fibrous tissue organization of both the external and internal coverings of the prosthesis is evident by the end of one month. This process of fibrous tissue organization continues rapidly and by 2½ to 3 months after implantation, connective tissue cells have invaded about three-fourths of the inner lining of the prosthesis. As time goes by, this fibrous connective tissue becomes hyalinized and on the inner surface the cells are flattened, resembling true endothelium. Thus, about 4 months are required to connect the internal and external fibrin deposits into fibrous connective tissue layers on the prosthesis.

Clinical studies of the morphologic changes occurring in and about synthetic prostheses after implantation have been made infrequently and at relatively short intervals after operation. The post-mortem findings in 2 cases studied 8 and 24 months, respectively, after operation were made available to the Committee and are as follows:

CASE 1.—A 55-year-old man was admitted to the hospital with an aneurysm of the abdominal aorta. The aneurysm was resected and replaced with a knitted Orlon bifurcation prosthesis. Subsequently, the prosthesis functioned well and about 6 months after operation

the patient developed symptoms of intestinal obstruction. At operation he was found to have a carcinoma of the sigmoid colon which was resected. Two months later (8 months after the implantation of the aortic prosthesis), the patient succumbed from extensive metastases of the colonic carcinoma. At necropsy, the aortic bifurcation prosthesis was completely covered with dense fibrous connective tissue so that the lines of anastomosis were not discernible; however, the prosthesis was soft and pliable (Fig. 6). The inner surface

Fig. 6. Fig. 7.

Fig. 6.—Photograph of a knitted Orlon prosthesis removed at necropsy 9 months after implantation; the patient died of carcinoma of the colon. The suture lines as well as the prosthesis are completely obscured by dense, fibrous, connective tissue.

Fig. 7.—The specimen shown in Fig. 6 has been opened longitudinally to show the inner surface of the prosthesis. There is a thin, transparent layer on the luminal surface of the prosthesis. The suture lines are well healed and the lumen is widely patent.

of the graft was covered with a thin transparent layer that was somewhat thicker at the lines of anastomosis than elsewhere (Fig. 7). Scattered over this inner layer were occasional flecks of fibrin clot but generally the surface was smooth. At the lines of anastomosis silk

sutures were plainly visible but completely covered by a thin membranous layer. Nowhere was wrinkling of the prosthesis evident and the lumen was widely patent. Microscopically, the inner lining of the prosthesis consisted of fibrous connective tissue which communicated through the interstices of the prosthesis with a similar fibrous connective tissue layer on the outside (Fig. 8). There were extensive infiltrations of inflammatory cells and giant cells of the foreign body type, particularly about the strands of yarn. At the anastomosis, union between the aorta and the prosthesis had taken place by means of dense fibrous connective tissue hyalinized in places. The inner lining continued across the suture line and on to the adjacent endothelial surface of the aorta and iliac arteries. Thus, as indicated from experimental studies, the porous synthetic vascular substitute provides a supporting frame upon which a fibrous connective tissue tube is formed. (In view of the known inherent weakness of hyalinized fibrous connective tissue to withstand continued stress, it is apparent that the synthetic yarn of which the prosthesis is constructed is the sole supportive structure in this reconstructed segment of aorta.)

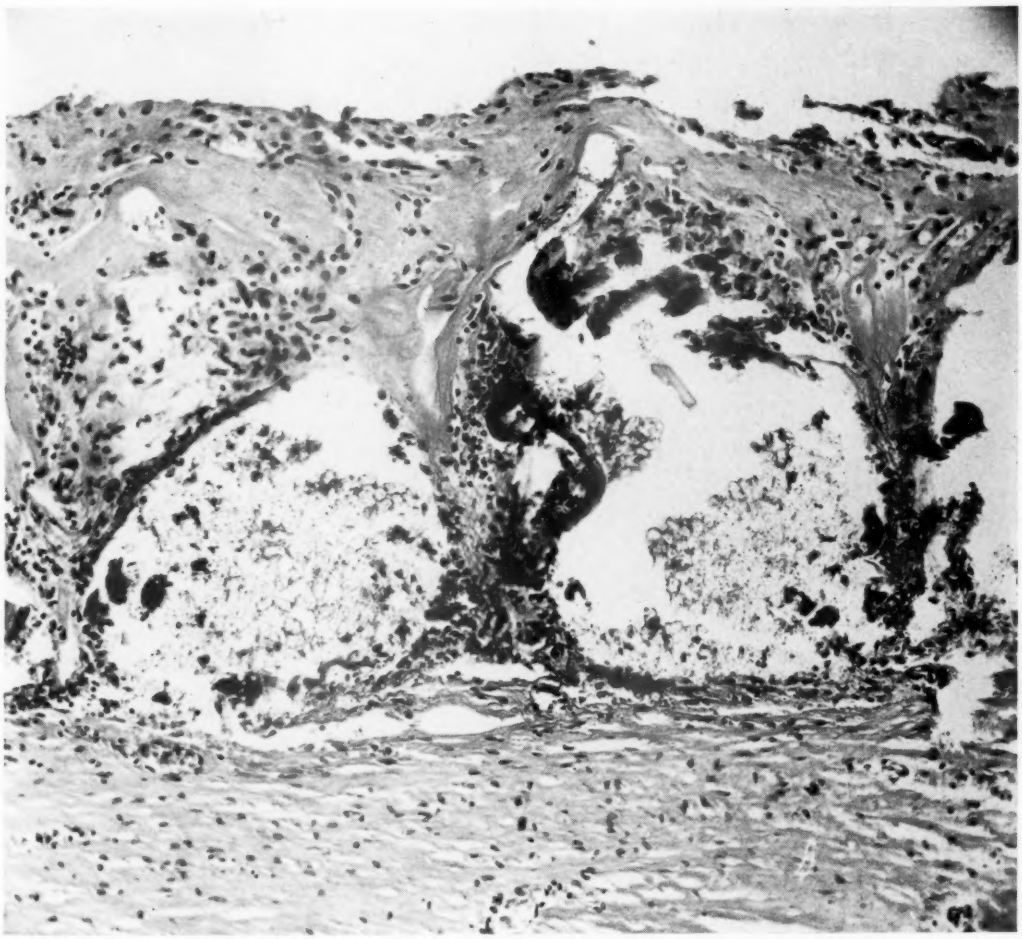


Fig. 8.—Photomicrograph of a section from the prosthesis shown in Fig. 7. The Orlon yarn is surrounded by dense fibrous connective tissue and an intense inflammatory reaction is present as evidenced by infiltrations of lymphocytes, plasma cells, and giant cells of the foreign body type.

Case 2.—A 65-year-old physician was admitted to the hospital for treatment of an aneurysm of the abdominal aorta. The aneurysm was resected and replaced with an Orlon cloth prosthesis treated on its external surface with liquid vinyl plastic to render it impervious. The fabric was a taffeta weave and was prepared by the surgeon on the sewing

Fig. 9.

Fig. 10.

Fig. 9.—An Orlon prosthesis coated with vinyl plastic removed at necropsy 2 years after implantation; the patient died from coronary occlusion. The prosthesis is completely encased in dense, hyalinizing, fibrous, connective tissue and there is acute angulation of the iliac branches.

Fig. 10.—The prosthesis shown in Fig. 9 has been opened longitudinally to show the inner surface which is covered by a thin layer of fibrin, disrupted in places by handling. Immediately outside the prosthesis there is an organized thrombus, and surrounding this is dense, fibrous, connective tissue. Although the iliac branches of the prosthesis are acutely kinked, the lumen is still patent and no thrombosis was evident in this area.

machine. The prosthesis was then placed on a brass mandrel, and the external surface was coated with vinyl plastic, and after air-drying for 24 hours it was implanted into the abdominal aorta. Two years after operation, during which time the prosthesis functioned well, the patient died from coronary thrombosis. At necropsy, the area of the abdominal aorta into which the prosthesis had been implanted was coated by a dense layer of fibrous connective tissue which completely obscured the prosthesis and adjacent aorta and iliac arteries. Upon removal of the aorta with the adjacent connective tissue and under careful dissection, it was found that the tissues surrounding the prosthesis consisted essentially of 2 layers, an inner layer of rather smooth glistening tissue and a thicker outer covering which was adherent to adjacent retroperitoneal structure (Fig. 9). Beneath the inner covering layer there was organized thrombus in places. The most striking observation was that of

Fig. 11.—Photomicrograph of the Orlon prosthesis shown in Figs. 9 and 10. The Orlon fibers appear as refractile bodies in the space in the center of the photograph. The prosthesis is covered on inner and outer surfaces by dense, hyalinizing, fibrous, connective tissue.

marked kinking of the iliac branches of the prosthesis about 2 cm. distal to the bifurcation. When opened longitudinally the prosthesis was found to be lined by a layer of fibrin about 1 mm. in thickness which extended across the suture lines on to the adjacent aorta and iliac arteries (Fig. 10). Although this layer was loosely attached to the inner surface of the prosthesis it appeared to be intact in all areas. At the site of kinking of the iliac branches of the prosthesis, the lumen was reduced by three-fourths, yet there was no thrombus evident either above or below the sites of constriction. The external covering of the prosthesis

literally fell away from the graft, and only at the lines of anastomosis was there any firm attachment of these tissues to the graft. Thus, grossly the prosthesis appeared to have been encysted by adjacent soft tissues. Microscopic sections were difficult to obtain because of the loose attachment between the prosthesis and surrounding tissues. However, it was possible to procure some sections which were suitable for study. These revealed the inner layer to be composed of fibrous connective tissue near the lines of anastomosis for about one-third of the distance toward the center of the prosthesis and fibrin, showing signs of early organization, in the center of the prosthesis. The outer surface was covered by dense hyalinizing fibrous connective tissue with many giant cells of the foreign body type and inflammatory cells. Anastomosis between the prosthesis and the adjacent aorta and iliac arteries was accomplished by fibrous connective tissue growing across the suture line both internally and externally (Fig. 11). However, a competent union between the two structures appeared to have taken place. Thus, except for the area of kinking the lumen was widely patent and a thin inner lining had formed which was similar in many respects to that noted in the pervious Orlon prosthesis previously described. Although the impervious substitute was not rigidly adherent to the tissues, there did not appear to be sufficient mobility to constitute a hazard to function.

PROPERTIES OF PROSTHESES

Each recipient of the questionnaire was queried regarding the properties that he considered important in a vascular prosthesis. The replies to this question are as follows, the properties being listed in order of frequency: permeable, flexible, easy to handle, inert, seamless, easily adaptable to recipient vessel, elastic, noncollapsible, and thin-walled. Almost without exception, the property of permeability was considered to be extremely important by those questioned. This was so because of the impression that the growth of fibrous connective tissue through the interstices of the material can affix the prosthesis to the tissues and at the same time speed up organization of the fibrin deposit on the inner surface of the prosthesis. Ease of handling is an obviously desirable quality. Inertness is another quality which is essential if the synthetic material is to be accepted by the recipient tissues and if the possibility of carcinogenesis is to be avoided. There were a number of workers in this field who did not believe the presence of a seam was detrimental provided it offered no impedence to blood flow and as long as the seams were well constructed and equally as strong as the prosthesis itself. The property of being easily adapted to the contour and diameters of the recipient vessel, although mentioned by only a few of those queried, was considered to be extremely important by those having the most experience with synthetic vascular substitutes. This was mainly because of the fact that these prostheses do not conform to a larger or smaller diameter of recipient vessel as readily as does the homograft. Therefore, the prosthesis must either be available in a variety of shapes and sizes or should be constructed of a material which will conform in a fashion similar to arterial homografts. There were few who considered the property of elasticity an important one, largely because of the fact that with the ingrowth of fibrous connective tissue, the distensibility of the prosthesis would be limited anyway.

DISCUSSION

The results of this study clearly indicate that vascular prostheses made of synthetic materials may be used effectively as aortic replacements. In fact, the reported 93 per cent successful results in 256 cases compare favorably with the

experience with homografts. From a functional standpoint synthetic vascular substitutes appear to be quite as satisfactory as homografts. Similarly, the acceptance of these materials by recipient tissues is little different from that observed with homografts, that is, both produce a moderate inflammatory reaction and eventually become incorporated into recipient tissues largely by growth of fibrous connective tissue. The ultimate morphologic appearance of synthetic prostheses or homografts has not yet been determined, but from a few observations made at 2 years, degenerative changes appear more likely to develop in homografts. Indeed, recent studies have demonstrated the development of typical atherosclerotic lesions in human aortic homografts from 1 to 2½ years after transplantation. Synthetic prostheses are more difficult technically to use and, as previously mentioned, are less adaptable to varying aortic diameters than are homografts. In addition, the pervious prostheses bleed momentarily until clotting occurs in the interstices, and this may constitute a serious problem as indicated by the report of 2 deaths resulting from uncontrollable hemorrhage through the walls of the prostheses.

Although experience with aortic substitutes is encouraging, it must be emphasized that the aorta is not a critical proving ground for vascular prostheses. The large diameter of the aorta and its major branches and the velocity of blood flow through them render the possibility of luminal occlusion from thrombosis unlikely irrespective of the types of material used. On the other hand, a peripheral artery, because of its relatively small size, great length, and crossing of flexion areas, must be replaced by a prosthesis more closely approximating in physical properties a natural artery if satisfactory function is to be obtained. Of all the materials investigated, none meet these criteria at the present time. For this reason the Committee feels that increasing emphasis should be placed on investigation of synthetic substitutes for peripheral arteries. To be sure, crimped nylon tubes and Dacron taffeta prostheses are being employed successfully in a number of instances; however, the follow-up period is relatively short and successful results have not been uniformly achieved.

In regard to properties of the ideal vascular prosthesis, the Committee was of the opinion that inertness, flexibility, ability to retain its shape, and ease of handling were all desirable qualities. Elasticity is probably not essential. The presence of seams is unimportant but the necessity for construction of seams as strong as the material itself is emphasized by the report of 2 instances of disruption of a prosthesis at the seams. In view of the results noted in one impervious aortic substitute examined 2 years after transplantation, it appears that additional experience with impervious materials is needed in order to determine the desirability of or necessity for a material through which connective tissue cells can grow.

Although synthetic vascular substitutes may be used successfully for aortic replacement, the durability of these materials under the biologic conditions imposed by transplantation has not been fully determined. On the basis of recent studies,* however, it appears that nylon retains no more than 20 to 25 per cent of its original tensile strength after periods of implantation of 7 to 18

*Capt. J. H. Harrison and Dr. William H. Moretz: Personal communication.

months (Table XVII). Since, in addition, the composition of nylon is extremely variable, this synthetic is probably less desirable for blood vessel prostheses than Dacron, Teflon, and Orlon. It is necessary to determine not only the tensile strength of the individual yarns and of the finished prostheses before and after varying periods of transplantation, but also the fatigue tolerance of the prostheses under simulated conditions of transplantation. Arrangements have been completed with a commercial laboratory for the investigation of these factors.

TABLE XVII. AVERAGE LOSS OF STRENGTH OF SYNTHETIC MATERIALS (INSTRON TEST OF ½ INCH STRIP)

MATERIAL	DAYS	PER CENT CHANGE
Teflon	150-200	+4.8
	301*	+10.2
Dacron	180	−4.7
	328	−3.6
Orlon	150-180	−12.8
Nylon	140-180	−80.1
	365	−81.0
	495	−80.0

*In pericardial defect.

In summary, it appears that synthetic replacements need have less critical characteristics for use in the aorta. Construction of prostheses by weaving, braiding, or knitting has been found satisfactory. The most satisfactory materials appear to be Dacron and Teflon at the present time. Vinyon-N cannot be autoclaved and is not made commercially as yarn. A significant loss in tensile strength has been observed in experimental aortic replacements of nylon and Orlon taffeta (Moretz, Poth, Harrison). A complete evaluation of Ivalon is not possible at the present time, but there have been more failures from rupture or thrombosis reported with this material than with all others. In peripheral vessels, a lesser degree of success has been achieved than in the aorta, although good function has been reported with taffetas of Dacron and nylon and with braids of nylon. For proper evaluation of replacement of peripheral vessels, the Committee plans a more detailed investigation of selection of patients and technique of implantation in cases to be reported in the future.

It must be emphasized that this is a preliminary report and the data presented here are not complete and do not represent the total experience with vascular prostheses. Undoubtedly, the experience of those failing to answer the questionnaire and those who were not questioned at all would be significant additions to the study. Furthermore, the results of studies now in progress may alter some of the conclusions reached in this report. Therefore, the Committee has been authorized to continue its investigations in this new and rapidly expanding field. Efforts should be directed toward standardization of materials and methods of construction for the protection of surgeon and patient. Improvement of existing prostheses should continue as well as the development of new substitutes, especially for the peripheral arteries. Since reasonably suitable replacements already exist, it is suggested that new ones be tested by independent investigators in animals for periods in excess of one year before trial in human patients.

INDICATIONS FOR SURGICAL TREATMENT OF THE POSTPHLEBITIC SYNDROME

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IN 1917, John Homans[5] was the first surgeon to recommend excision and grafting of postphlebitic ulcers. Twenty years later, he wrote on the treatment of deep thrombophlebitis: "There should be urged, when the leg appears normal, the application of a semi-elastic bandage and guarded exercise in the standing position. First, a few minutes of exercise followed by elevations; the periods of exercise being gradually prolonged if the leg does not swell or become cyanotic. . . . If no recurrence follows, you can call the patient cured. Otherwise, the femoral vein is divided distal to the profunda."[7]

Many surgeons have since shown more interest in the surgical treatment than in Homans' original description of progressive ambulation. Nearly thirty operations have been devised. One surgeon may advocate ligation and partial excision of a deep vein,[1, 10] while another develops a valve for this vein.[4] The superficial veins may be excised along with the surrounding scar tissue to remove varicose or occluded venous and lymphatic channels, [8, 14] yet others inject veins and consequently occlude adjacent lymphatics.[15, 16] Paradoxically, a surgical excision produces scar tissue, and surely the exact amount to excise cannot be determined without a radical procedure, which again has many advocates. Modifications include removing all of the skin, subcutaneous tissues, and fascia, followed by skin grafts[6] or excising the underlying tissues and leaving the skin intact.[10] More diversification appears when the advocates of sympathectomy maintain that vasospasm needs to be relieved to increase the blood supply.[14] Yet another procedure suggests that the limb has too much arterial blood, and it is necessary to narrow the peripheral arteries to decrease this flow.[3] But reported good results are not duplicated at other centers, and no definitive answer to the problem of surgical therapy emerges.

Having had varied results with most of these and other well-known surgical procedures, we decided in 1952 to initiate a thorough conservative plan of therapy which we hoped would assist us in establishing accurate indications for surgical treatment. Our results have been so gratifying to date that surgery has played a very minor role in the care of these unfortunate individuals.

During this 4-year period, 154 patients in the indigent or low-income groups were treated. Although the period of observation of some patients is short (minimum 6 months), the majority were observed long enough that we believe we have adequate information to support our conclusions.

Read at tenth annual meeting of the Society for Vascular Surgery, Chicago, Ill., June 10, 1956.

DIAGNOSIS

Diagnosis of the postphlebitic syndrome in the 154 cases was based on a history of deep vein thrombophlebitis followed by edema and any combination of skin pigmentation, dermatitis, subcutaneous fibrosis, chronic ulceration, varicose veins, recurrent infection, and such disabling subjective symptoms as heaviness, fatigue, and pain. In some cases without history of thrombophlebitis, the typical findings of the syndrome were present, but no other known cause (e.g., cardiac disease, primary lymphatic obstruction, simple varicose veins) could be found; these were considered nonthrombotic.[1] This group numbered 23 (17 per cent), while 129 cases (83 per cent) showed classical evidence or history placing them in the thrombotic group.[10]

REVIEW OF CASES

Female patients, as expected, outnumbered male patients 93 (61 per cent) to 61 (39 per cent). Ages ranged from 17 to 86 years, with averages of 61.3 years for the men and 54.9 for the women.

Only 18 of the entire series (12 per cent) required admission to the hospital for treatment. They were admitted because of recurrent thrombophlebitis, cardiac failure, marked cellulitis of the extremity, malignant or large unresponsive ulcers, necessity for excision and ligation of varicose veins, or necessity for grafting of ulcers.

Duration of the postphlebitic syndrome ranged from less than 6 months to 53 years, with an average of 10.3 years.

Lower extremity edema (especially morning) was present in all patients, 91 (59 per cent) being unilateral and 63 (41 per cent) bilateral. Few patients had edema as the only presenting symptom or sign. Variables of dermatitis, pigmentation, varicose veins, and the like accompanied the edema. No ulcer was present in 69 cases (45 per cent) while 60 (39 per cent) had unilateral ulcers and 25 (16 per cent) showed ulcers bilaterally. The duration of these ulcers averaged 7 years, while the syndrome without ulcer lasted for an average of 9.3 years. The shortest period in which an ulcer appeared was 6 months; the longest was 38 years. Trauma was not the cause of ulceration in all cases. In some instances, a traumatic ulcer formed first, then the syndrome reappeared and remained. In other instances, the edema returned as a result of pregnancy, cardiac failure, or recurrent thrombophlebitis; the previously thrombosed extremity again manifested edema, and an ulcer formed.

Three female patients (2 per cent) were admitted with malignant ulcers showing squamous cell carcinoma. Each ulcer had been present more than 30 years, although one of them had healed for a short period 17 years previously. There was no history or clinical evidence of osteomyelitis in two of these cases, but one patient developed bone involvement during the terminal year.

Fibrosis about the Achilles' tendon resulting in an equinus deformity, a seldom reported complication of the postphlebitic syndrome, occurred in 2 female patients with ulcers (1 per cent).

No major varicosities or incompetency of the long or short saphenous system were observed in 49 cases (32 per cent) or 140 extremities. Minor isolated varices were not classified. While 105 patients (68 per cent) or 168 extremities showed some major component incompetent, 56 of these were present before the syndrome developed and 49 appeared after its onset.

No allied disorders were noted in history or on examination in 33 cases (21 per cent) while 121 patients had some allied condition which could cause a flare-up of the syndrome or hamper therapy. Careful histories and physical examinations were done to be sure that these conditions were not causing the edema. The most frequent disorders were (1) obesity, (2) cardiovascular conditions including cardiac failure, cor pulmonale, hypertensive cardiovascular disease, and arteriosclerosis obliterans, and (3) mental disease. Multiple disorders were frequently present.

A surprising number of operative procedures had been done before these patients first visited our clinic—81 on a total of 124 extremities. Sclerosing therapy by injection had been done on 35 patients or 61 extremities, some patients having had more than 50 injections. Six patients developed marked dermatitis or reappearance of ulcer, and 3 who had never had deep vein thrombophlebitis developed the postphlebitic syndrome following such therapy.

TABLE I. ALLIED DISORDERS

No allied disorders		33
With allied disorders		121
Obesity	44	
Cardiovascular	42	
Mental	30	
Metabolic	15	
Syphilis	10	
Malignancy	9	
Arthritis	5	
Pelvic inflammatory disease	4	
Primary anemia	3	
Genitourinary	3	
Blindness	2	
Total patients		154

Note: Numerous patients had more than one of the above disorders.

TABLE II. PROCEDURES PERFORMED PRIOR TO PRESENT THERAPY

	PATIENTS	EXTREMITIES
Injections of veins	35	61
Skin grafts	10	18
Superficial femoral vein ligations	4	6
Inferior vena cava ligations	6	–
Lumbar sympathectomies	3	3
Excision, ligation, or stripping varicose veins	23	36
Total	81	124

Fifty-six surgical procedures on 67 extremities made up the remainder. Ten were skin grafts to ulcers on 18 limbs, 3 having had 2 grafts to the same ulcer, and 1 having had a recurring ulcer grafted 4 times. Superficial femoral vein ligations had been performed on 4 patients (6 extremities) and 3 lumbar

sympathectomies had been done. All had been unsuccessful for their indications. Twenty-three patients had had excision, ligation, or stripping of varicosed veins on 36 limbs. In addition, 6 inferior vena caval ligations had been done for thromboembolism and were followed by the syndrome.

Surprisingly, only 25 patients of the 129 in the thrombotic group had had anticoagulants. However, 19 additional patients were classified as "clotters," 6 of whom needed total protection on long-standing anticoagulant therapy.

TREATMENT

Treatment was established principally on an outpatient basis, following a complete general medical examination. Each of our vascular clinics (Colorado General and Denver General Hospitals) is staffed by a team consisting of an internist, a surgeon, and a chiropodist. Interpreters, clinic or visiting nurses, and members of the family are often called upon to aid in the education of the patient.

The internist and the surgeon see every patient jointly to evaluate the extremity involved and the patient's over-all problem. The team first initiates a conservative treatment program consisting of (1) patient cooperation, (2) treatment of allied disorders, (3) control of infection, and (4) elevation, compression, and exercise of the extremity.

Patient cooperation is demanded for thorough education in the physiopathology of the condition, both for immediate care and for continuing prophylaxis. Mimeographed instruction sheets are issued, and the patient is repeatedly "quizzed" on this material. Cooperation implies not only compliance with instructions for lower limb care, but also willingness to assist in caring for allied disorders.

Allied disorders, as listed in Table I, were present in 121 cases; we emphasize the importance of "total" treatment. Those with cardiac failure may require digitalization, diuretics, and salt-poor diets. Obesity, of course, necessitates weight reduction. Thrombophlebitis when still active or recurrent is treated on an inpatient anticoagulant basis until there is complete subsidence of symptoms. No effort is spared in obtaining successful treatment of the allied disorder, since this is prerequisite to successful primary treatment of the postphlebitic syndrome.

Elimination of infection, whether it be dermatitis, epidermophytosis, or infected ulcer, is approached with the concept that the majority are due to fungi. Therefore, treatment is initiated by means of potassium permanganate soaks at home three times daily. When an ulcer appears to be infected by bacteria or fungicidal treatment is not adequate, antibiotic ointments such as bacitracin and neomycin are applied to the ulcer several times a day. When cellulitis is not present and frequent applications of medication may be harmful, an Unna boot is used for a 3-week period. Occasionally when an ulcer and surrounding skin have become macerated, we employ agents such as gentian violet or 20 per cent Mercurochrome to bring about drying, being careful that no infection pockets beneath the eschar. We do not give parenteral penicillin unless the patient has such a widespread cellulitis that admission to the hospital is warranted.

The edema is initially cleared by elevation. This is usually accomplished overnight, but some patients required as much as 2 weeks of continuous elevation to reach the edema-free state. Active and passive exercise during elevation was encouraged especially in those patients whose edema was slow in subsiding. Progressive ambulation was begun using elastic compression support only after the edema had disappeared.

Four types of elastic compression support have been used. If the leg was markedly swollen and edema returned on ambulation in less than one hour or if previous therapy with cloth-rubber support had failed, pure rubber (Esmarch) bandages were advised; a cotton stocking may be worn beneath this to avoid skin reaction to the rubber. Elastoplast, as advised by Linton,[10] was used with similar indications. Most cases, however, require only cloth-rubber compression from the beginning. Following an increase to 4 or more hours of edema-free ambulation, cloth-rubber compression bandages are substituted for the rubber bandages. Elastic stockings, unless tailored, do not appear to be a wise choice for patients having morning swelling and are ordered only after morning edema has been controlled. In no case was the elastic support worn above the knee.

Active ambulatory time was stopped just short of the appearance of fullness, heaviness, fatigue, or edema in the extremity, and the patient returned to elevation for at least a 15-minute period. Each day the period of ambulation was increased short of the expected appearance of the fullness or edema. These periods may fluctuate with the type of exercise, and the patient is so informed. In general, each week finds improvement; if not, the patient's habits and daily activity are reviewed to find the reason for failure. When he has reached a 2-hour edema-free period (usually in less than 2 weeks), the patient is allowed to return to work, provided he may elevate the extremity (above heart level) for a 15-minute period at the end of the 2 hours. This is then extended to four, then six, and finally left to the patient's discretion. By this time, he is wearing an elastic stocking, and before long most patients will be able to go without support most of the time. Sitting or standing for long periods is discouraged during early therapy unless active movement of the extremity is undertaken—an attempt to utilize the "peripheral heart."

In summary, the patient is instructed that he must "never be ambulatory when fullness or swelling is present, or without his elastic support," and each case is individualized throughout the treatment. Foot care is stressed and prophylactic medications are prescribed; talcum and fungicidal powder for the moist foot or leg wearing an elastic support, and lanolin or an ointment with a methyl cellulose base for the dry, scarred, or indurated extremity. Our maxim for foot hygiene is "never wash your face without washing your feet." Corns, callouses, thickened toenails, and the like are managed by the chiropodist assigned to the clinic.

Certain *operative conditions* which appeared early in the program needed correction to bring about a successful outcome. Indolent ulcers may need grafting, or immediate healing is sometimes required for economic reasons. Seldom does the latter present a problem, for most ulcers heal within 4 to 6

weeks no matter what is applied to their surface, providing the fungus infection is cleared and the edema is controlled. However, wide excision and grafting are advised if this healing is delayed or if the ulcer has been present for several years and malignant degeneration could possibly be present. When malignant lesions do develop, it will be necessary to do wide excision and grafting and possibly even amputation or radical groin dissection when palpable nodes are present.

Two factors are important in treatment to prevent the complication of Achilles' tendon shortening causing an equinus deformity, which occurred in two of our cases: (1) when an ulcer is present, it is inadvisable to wear high-heeled shoes for relief of pain; (2) prompt healing is imperative, for such an ulcer overlying the tendon presents a problem in grafting. It may be necessary for these patients to sleep with posterior ankle splints. Delay in therapy may require future tenoplasty.

Large, brawny ulcers with surrounding induration and edema failing to respond to elevation-exercise after 2 to 4 weeks should be considered for radical excision, either a modified Kondoleon[6, 9] or Linton's[10] similar procedure. Those patients with persistent edema, with or without ulcer, may also be considered in this category.

The patient with a cold, sweating, superficially painful, cyanotic or pale foot of the causalgic type is given an intra-arterial vasodilator first, which not only establishes the diagnosis but also will sometimes give permanent relief. This is followed, when indicated, by oral vasodilators, paravertebral lumbar sympathetic blocks, or, if no permanent relief occurs, by lumbar sympathectomy. We do not believe the postphlebitic syndrome without the above signs and symptoms is an indication for sympathectomy.

Varicose veins are not approached surgically until the patient has had 3 to 6 months of conservative therapy. Even then, this is delayed until the morning edema has been controlled. We have excised and stripped these veins in stages on an outpatient basis in the operating room, using either epidural or local anesthetic; we advise hospitalization only if the case has been difficult to treat because of allied disorders. We instruct the patient to walk with elastic support to the knee 10 minutes out of every hour for the few days following operation and to follow the postphlebitic syndrome routine. The afternoon swelling which may have been present before surgery usually disappears, and the patient wears a cloth-elastic bandage or stocking for about 3 months. When no swelling appears during the day, he may ambulate without support. He is warned that any trauma or infection to the extremity may result in return of the syndrome and necessitate return to the elevation-exercise routine.

RESULTS

Excellent results (no further treatment or clinic visits necessary) were obtained in 69 cases (45 per cent), as determined by follow-up from 6 months to almost 4 years. Good results (occasional slight swelling and clinic visit

every 3 to 12 months for minimal additional advice) were observed in 61 cases (40 per cent). Combining these two groups gives a percentage of 85 per cent satisfactory results.

Fair results (occasional swelling and needing frequent clinic visits) were reported in 13 patients (8 per cent). Poor results (minimal response to therapy and recurrence of ulcer or failure to heal an ulcer) were obtained in only 11 cases (7 per cent).

Reasons for treatment failure were sometimes multiple. There were 55 allied disorders in the 24 patients with fair or poor results. Fifteen of the 24 patients were uncooperative in failing to attend clinic regularly, refusing to accept personal responsibility, or showing mental imbalance or low intelligence, alcoholism, or drug addiction. Nine of these 24 patients were obese, and 6 of these were uncooperative in other ways than failing to lose weight. Eight uncooperative mental patients had fair or poor results. Six had had malignant lesions, such as carcinoma of the breast, ovary, or cervix, excised previously with no metastases noted; 2 of these had malignant ulcers (squamous cell carcinoma) forming in 30-year postphlebitic ulcers, which necessitated amputation. Recurrent infections and ulcer or thrombophlebitis occurred in 8 cases; 2 had recurrent ulcers after previous healing. Two primary anemias (pernicious and aplastic) were in the poor outcome group. Only 1 patient was advised to change his job; since he has been unable to do so, he still needs frequent checkups. Even though 42 patients were noted to have some type of cardiovascular disorder, only 3 of these were listed in the poor results group after therapy. The duration of edema or ulcer did not seem to influence results except in 2 of the 3 malignant ulcers.

TABLE III. RESULTS OF THERAPY
(154 PATIENTS)

Excellent (No further treatment necessary)	69	85 per cent
Good (Occasional slight swelling; ulcer healed)	61	
Fair (Swelling necessitating frequent checkups)	13	15 per cent
Poor (Recurrent ulcer or persistent swelling)	11	

In this series, 28 patients were operated upon, including 37 extremities. Most of these were excision and stripping of varicose veins done 3 to 6 months after initiation of treatment and not until swelling had completely disappeared or had been lessened to afternoon swelling. Three patients had ulcers excised and skin grafts applied early in the program; none have recurred. One malignant ulcer was widely excised and grafted; no recurrence has been noted in the 18-month follow-up. One modified Kondoleon was done 3 years ago for a large maggot-filled ulcer on a brawny, edematous leg which failed to benefit from 6 weeks of therapy; the patient is much improved, but still needs fairly frequent follow-up and observation for recurrent epidermophytosis.

One patient classified in the fair group had a plication of the superficial femoral vein producing a venous valve as described by Eiseman.[4] No change has been noted in his status. He has not been off work except for two hospitalizations (valve and recurrent thrombophlebitis) and controls his edema by wearing high-topped laced boots while working in a frozen food storage establishment.

Allied disorders in all cases except the failures listed have been cured or improved sufficiently to aid in the improvement of the syndrome.

The 2 cases with Achilles' tendon involvement and equinus deformity were seen early and cleared after healing of the ulcer and disappearance of the edema. High-heeled shoes were not worn until all active pathology had subsided.

Average age of our patients is probably high in relation to other reports since no private cases have been recorded and the survey includes only members of the indigent and low-income groups. Many younger patients seen early in their syndrome have returned to work and are being followed by their local physicians. These patients have been lost to follow-up and are not listed.

The percentage of cases handled on an outpatient basis (88 per cent) reduces the expense of inpatient care, and the 2-hour edema-free rule for return to work decreases the number of man-hours lost to industry. In only one case have we advised change of occupation. We have made every effort to fit the therapy into the patients' daily routine by means of the type of bandage and utilization of the American custom of "coffee breaks" and the lunch hour—the 2-hour intervals between beginning of the workday, coffee break, lunch hour, coffee break, and the end of the day.

Insistence that the extremity be edema-free before ambulation is started is a sound and well-established principle allowing nature to develop collaterals without hindrance. Major veins recanalize and conceivably delay the recovery period, but this recanalization may only cause additional harm when collaterals have not been protected during and after their development. Choking of these vessels from beneath the integument by edematous fluid can only cause harm in this process.

Clearing of ulcers has not generally been a problem once edema and fungus contamination are removed. Large ulcers on a brawny, indurated extremity and the rare malignant ulcers are definitely the biggest problem in this group. Of the 8 patients whose edema did not subside after prolonged elevation, only 1 needed radical therapy; the remaining 7 are much improved, but not enough to be recorded in the satisfactory results group. Two of these would be candidates for radical therapy, while the other 5 have other disorders contraindicating any major procedure. Continued education keeps them under satisfactory control.

We have had more than our share of malignant ulcers related to the postphlebitic syndrome. Few have been reported in the literature—Martorell[13] had only 1 in 769 ulcers—but in treating long-standing ulcers, doctors should be aware of this possibility.

TABLE IV. PROCEDURES DURING PRESENT THERAPY

	PATIENTS	EXTREMITIES
Excision and stripping of varicose veins	22	31
Skin graft	4	4
Modified Kondoleon	1	1
Valve (plication superficial femoral vein)	1	1
Total	28	37

Varicosities frequently bring out complaints which appear exaggerated, even in patients without the postphlebitic syndrome. Afternoon and evening swelling is not uncommon in these patients. The postphlebitic with varicose veins should be spared this potential source of edema, which is frequently resistant to therapy after morning edema has cleared. Removal of these damaged veins almost invariably brings clearance of this residual. We are strongly against sclerosing therapy, especially when a concomitant postphlebitic syndrome exists.

Venography for study of the deep venous system was discontinued in our clinic about 5 years ago, when we concluded that complications do occur, findings were difficult to interpret accurately, and the procedure was more of academic interest than of specific benefit to the patient.

Resistant edema, especially in the thigh, has frequently been relieved by means of parenteral or oral diuretics. We have occasionally used intramuscular hyaluronidase with success. Intramuscular trypsin has been used, but not enough for us to feel qualified to advise its use routinely. When thigh edema remains, the patient should be ordered to bed for evaluation-exercise until edema-free.

There is general agreement regarding the initial treatment of acute thrombophlebitis by means of rest, elevation, sympathetic denervation, anticoagulants, antibiotics with infection, and possibly major vein ligation when pulmonary emboli occur. In addition, attention is directed toward establishing a diagnosis and etiology and toward prophylaxis of deep vein thrombosis. Therefore, we need to treat the postphlebitic syndrome from the same broad viewpoint as we do the acute disease.

We agree with recent editorials[2, 12, 15] suggesting a pooling of long-term results of therapy by the many clinical investigators to determine what method is best for these patients—whether it be conservative, surgical, or a combination of the two. We emphasize that therapy should be wisely chosen, and specific indications for surgery must be considered.

This report follows Luke's[11] "new way of life" concept to a degree, but we do not feel that restrictions necessitate any radical change in the patient's activity for the future. It is true that an occasional case will be more complicated than average, and such a patient may find it necessary to change his occupation, restrict his daily activity, and/or receive continuous anticoagulant therapy. These may be the cases which warrant surgical intervention.

CONCLUSIONS

Conservative treatment gives 85 per cent satisfactory results in 154 patients with postphlebitic syndrome. This program requires careful patient education and patient cooperation, vigorous treatment of allied disorders,

control of local infection, and elevation, compression, and exercise followed by progressive edema-free ambulation with compression. Unsatisfactory results occur in failure to meet this program.

Our indications for surgery have been few. We have operated upon only 27 of these 154 patients. Reasons for operation have been: (1) An occasional graft for indolent ulcer which fails to heal within a 3- to 4-month period, especially if malignancy is questioned because of its appearance or extremely long duration. (2) Brawny, indurated legs with or without large unresponsive ulcers which have not shown subsidence of the edema in one month. These necessitate radical excision. In our series, they are uncommon. (3) Excision of varicose veins, our most common operation in this series, which was done for symptomatic relief only when the postphlebitic edema had been controlled for a significant period.

In view of these findings, we feel that surgery has its place, but should be reserved for those complications which have failed to clear after a thorough conservative regimen.

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DISCUSSION

DR. THOMAS T. MYERS (Rochester, Minnesota).—I certainly complement Dr. Owens on his excellent presentation of the problem. He has shown the great necessity for meticulous care of this type of patient.

I definitely consider elastic support important. The patient cannot get along without it. Most of us are convinced that the surgical approach to this problem is certainly not definitive. Ligation of the deep veins has not done what we expected it to do. Elastic support alone has not done what we hoped it would do. So we must have some other plan.

In my experience the superficial veins are an extremely important part of this problem. (Slide.) The stasis changes in the lower extremity all occur above the deep fascia. A stasis ulcer never penetrates the deep fascia. Commonly the cutaneous changes appear over the veins where they come through the fascia or close to that site. The femoral vein is behind the deep fascia all through its course. The small saphenous vein is behind good fascia. Its incompetency does not cause changes in the skin until a tributary becomes incompetent above the fascia. This commonly occurs in the calf where there is a tributary and frequently a vein that penetrates the fascia. The great saphenous vein is above the deep fascia throughout its course; thus it has much opportunity to cause changes in the subcutaneous fat and the overlying skin. Fortunately, these usually occur only from the knee distally. In the postphlebitic leg these changes are greatly exaggerated because the relatively increased pressure in the deep veins is transmitted above the fascia through the varices into the subcutaneous tissue and skin. An incompetent perforating vein increases this change greatly.

What can be done to control congestion of the subcutaneous tissue? In my experience adequate elastic support is essential in the advanced condition but removal of the superficial veins, if these veins are varicose, adds much to the ease of control. (Slide) This slide indicates that the operative treatment must be extensive and thorough. In some of our early cases only stripping to the knee was done. When results were compared with those of stripping throughout the lower extremity, partial stripping gave excellent and good results in only 58 per cent of cases and complete stripping gave excellent and good results in 94 per cent.

The incompetent perforating veins also are extremely important to the problem of congestion of subcutaneous tissues because they transmit congestion above the fascia. They must be ligated behind the fascia.

I wish to emphasize, too, that surgery of superficial veins if done properly is a great adjunct to the control of complications in the postphlebitic leg.

DR. GEZA de TAKATS (Chicago, Ill.).—I would like to inject a thought here which has gradually crystallized in our minds.

We started our interest in the postphlebitic syndrome in 1925 at the Vascular Clinic of Northwestern University, later transferred to Illinois. There is undoubtedly a group of cases, possibly 40 per cent of the patients, who have a well-compensated venous circulation. They have had either a good history of venous thrombosis or a vague history of it or no history of it at all, but the appearance of the venous pattern is definitely that of a collateral supply. Now, these patients, about 40 per cent of them, are doing very well. They have a slightly enlarged lower extremity. They have large varicosities. They have no pain. They need no treatment.

I have one of these myself and I would be very glad to stand up on that table and demonstrate it to you. I wear no hose and stand eight hours in the operating room at times. This, of course, has absolutely nothing to do with a decompensated venous circulation in a man or woman with a large amount of postphlebitic induration requiring large, split-thickness grafts or one who has recurrent attacks of phlebitis because of intercurrent infections.

While I wish to compliment Dr. Owens on this very sound, conservative attitude he has taken and while we have always recognized the great importance of compression, I do not think we should abandon surgical procedures. I agree with Dr. Myers on paying attention to the superficial circulation and it certainly is very important. A wide amount should be excised before grafting with the indurations being excised under the skin with the fascia, leaving the skin intact. The search for perforators and their ligation is mandatory.

But most important, as Dr. Owens said, is that each case in an individual problem. There is no absolute cure of this syndrome, anyway, as long as man will maintain his erect position, but while the tendency toward conservatism is justified it is not right to say that surgery is entirely useless and should only be limited to cases suspected of malignant degeneration. That to me is a step in the wrong direction.

DR. JOHN J. CRANLEY (Cincinnati, Ohio).—We agree that the postphlebitic ulcer can be healed while a patient is ambulatory in the majority of cases. In private practice 89 per cent of our patients have healed in this manner and in the outpatient clinic of the Cincinnati General Hospital approximately 75 per cent of the ulcers have been healed while ambulatory. Despite all our efforts to keep the leg healed with conservative measures, the recurrence rate is approximately 30 per cent. When, however, a radical vein operation is done as described by Linton the recurrence rate is exceedingly low. To date with the 4-year follow-up we have had one recurrent ulceration in approximately 90 extremities.

Kondoleon was treating lymphedema and made no mention whatever of veins in the description of his operation. Therefore, in our opinion his name should not be used in connection with this radical procedure we do today which centers entirely on incompetent veins.

Finally, one must choose between a basic philosophy of attempting to change the patient's way of life to suit his legs or to change his legs to suit his usual way of life. We strive to do the latter and believe it can be done successfully if sufficient time and care are taken to remove as many of the diseased veins as is possible and control swelling of the leg by a heavy-weight elastic stocking.

DR. THOMAS C. MOORE (Muncie, Ind.).—I too wish to compliment Drs. Owens and Anderson on their excellent study. This subject never fails to incite considerable comment.

Dr. Shumacker and I, from our clinical and our functional venographic studies, agree with the general approach to this problem suggested here by Dr. Owens. We feel that the management of the postphlebitic state is essentially nonoperative and that it should be directed largely toward securing adequate compressive support for the dependent extremity. Careful instruction concerning the avoidance of prolonged lower extremity dependency also is of importance. This recently has been emphasized by Luke and others.

I should like to limit my comments to the nature of the compressive support to be applied. Good compressive support is quite desirable and the most effective types of elastic stocking seldom can be obtained across the counter at the corner drugstore. The type of elastic support I use most frequently is the heavy-weight nylon elastic stocking manufactured by the Truform Company of Cincinnati, Ohio. I have been pleased with this means of support in the majority of cases and am indebted to Dr. Cranley for suggesting its use.

However, the real "ace in the hole" and the most effective of all of the types of compressive support is the Aero-pulse legging developed by Dr. Merle Scott. It is manufactured by the Surgical Research Corporation of Albion, New York. It is somewhat more expensive than the elastic type of stocking and is considerably more bulky. Its bulk renders it unsightly, especially for women. Nonetheless, when all other means of support fail, this excellent method of achieving good compressive support can be counted upon to do the job.

DR. ROBERT LINTON (Brookline, Mass.).—I would like to congratulate Dr. Owens on his excellent results with the conservative treatment in the postthrombotic syndrome. It is my opinion, however, that it is important to differentiate between the patient who has a postthrombotic syndrome without ulceration from one with ulceration. I personally believe that the former should be treated as Dr. Owens has described, but if an actual ulceration is present this should first be healed by conservative means and then later a radical operative procedure carried out after the ulceration is completely healed.

The method of treatment described by Dr. Owens gives these patients a great deal of relief, especially since it reconstitutes the function of the venous heart of the lower extremity by preventing the regurgitation of blood through the communicating veins into the superficial veins. In the nonulcerated extremity, to obtain the same result, I use a special type of two-way stretch, heavy-weight elastic stocking.

In many instances where ulceration is present, this method is not sufficient so that a radical operative procedure, in my hands, has proved the most effective method of instituting a cure. This consists of interrupting the deep venous circulation by ligation and division of the superficial femoral vein, ligation and division of the communicating veins on the inner and posterior side of the lower leg, and radical removal of the long and short saphenous veins and many of their tributaries. Unless this radical surgical method is carried out, it is better to treat the patient as Dr. Owens has described, since an incomplete operation will do very little, if any, good. It is well to remember and important to point out that the one thrombotic sequela that cannot be cured by surgical means is chronic lymphedema, and it is for that reason, even with this radical surgical procedure, I recommend the patient wear more or less indefinitely an adequate elastic stocking.

DR. J. CUTHBERT OWENS (closing).—I wish to thank the discussers for their interesting comments. I believe we are very close to agreement; however, I must frankly admit that before I started this afternoon I didn't believe this would be true.

We agree with Dr. Myers that varicose veins present in a postphlebitic syndrome should be removed, although as stated not until the syndrome is under control. We plan to excise the varices on the remainder of our patients where indicated. Our approach has been by excision under direct vision instead of a blind stripping technique. This lessens so-called recurrences, which we believe are, on the whole, "missed" varices. As we all know, varicose veins are prone to marked variation; therefore, patience plus surgical curiosity at operation are required to eliminate these segments.

I want to emphasize again that allied disorders are frequent with this condition, as with acute thrombophlebitis, and must be considered in the total treatment for a successful outcome. We have found this frequently lacking in published reports.

It is agreed that Kondoleon's original description of radical excision of tissue in the chronically edematous leg concerned lymphedema. Credit should be given to Dr. Homans for our present procedure of radical excision of edematous tissue in the postphlebitic syndrome. So Dr. Homans receives credit not only for the radical therapy, but also for conservatism. It is our belief that very few need radical therapy, while all need the thorough conservative program.

PRESIDENT GEORGE D. LILLY (Miami, Fla.).—Dr. Owens, I apologize for discussing your paper from the Chair, but I should like to make a comment as to the advice which can be given these people regarding home management.

Usually, these individuals will get out of bed, shave and bathe, and have breakfast before applying their elastic supports. I feel that it is very important that they adopt a routine of bathing in the evening, and that they apply the elastic support as they get out of bed in the morning rather than after they have been in an upright position for some time.

All of the present-day elastic stockings lose their elasticity rapidly, and due to their expense the patient is prone to wear them after they have become loose. As a result, insufficient pressure is applied in most cases. The pneumatic stocking is not satisfactory in warmer climates because it is airtight and does not permit evaporation. I have found that it is quite simple to apply adequate pressure and use stockings for long periods of time by reinforcing the stocking with a half length of elastic bandage; in other words, the patients uses the stocking as an inner lining and the elastic bandage to apply additional pressure. This is much more comfortable than the bandage by itself, because the stocking prevents the creasing and cutting into the skin that occurs with a bandage alone.

OBSERVATIONS ON PATIENTS DURING THE SURGICAL TREATMENT OF ACUTE MASSIVE HEMORRHAGE FROM ESOPHAGEAL VARICES SECONDARY TO CIRRHOSIS OF THE LIVER

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IT IS the consensus of surgical opinion that patients with portal hypertension and esophageal varices are best treated by some form of decompression of the portal circulation, either portacaval or splenorenal anastomosis. Even though it is admitted generally that the therapy does not alter the course of the disease,[3] some workers have gone so far as to suggest that portacaval anastomosis done as a prophylactic procedure will salvage a certain percentage of cirrhotic patients from the dangers of hemorrhage for a period of time.[17]

The decision as to proper therapy is even more difficult when one is called upon to treat the cirrhotic patient who presents with massive gastrointestinal hemorrhage. In an effort to provide some sort of answer to this most distressing problem, we have attempted to treat these patients by immediate ligation of the bleeding varix, as had been suggested by Boerema, Linton and Warren, Crile, and most recently Linton and Ellis.[5, 10, 11]

It is the purpose of this report to discuss some of the difficulties encountered in 23 patients operated upon during the acute stage of hemorrhage and to record some interesting physiologic data which we have accumulated.

The experience with cirrhosis of the liver is extremely bad at the San Francisco County Hospital. During the years 1930 to 1950, there have been 538 patients with this diagnosis in the hospital. As of 1951, aside from 97 patients lost to follow-up, only 44 were alive. One hundred and twenty-eight of these patients had died following a massive hemorrhage.

TABLE I. CAUSES OF GASTROINTESTINAL HEMORRHAGE IN CIRRHOTICS PROVED AT NECROPSY

Bleeding esophageal varix	38
No anatomical cause	23
Peptic ulcer	3
Multiple erosions	2
Esophagitis	2

The diagnosis of esophageal varices as the source of hemorrhage in the cirrhotic patient is by no means certain unless one is fortunate enough to have had a previous gastrointestinal x-ray examination. In Table I, the data on 68 cirrhotic patients who died from a massive hemorrhage are shown.

Aided in part by Grant A239, USPHS.

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The ever-present possibility that the hemorrhage is due to a lesion other than the varix has been noted by many writers on this subject.[6, 14, 15] In most series peptic ulceration occurs in about 15 to 20 per cent of patients with cirrhosis. It is for this reason that we have preferred to explore the patient through a high midline abdominal incision. The stomach is examined first. If no obvious ulcer is noted, the stomach is opened wide on the greater curvature, and the duodenum and fundus of the stomach are inspected after evacuating the blood from the stomach. If it is apparent that the bleeding is coming from the esophagus, the abdominal incision is extended upward, either splitting the sternum or cutting the cartilages to the left of it so that the distal 10 cm. of the esophagus can be exposed easily without cutting through the diaphragmatic portion of the esophagus. A horizontal incision then is made

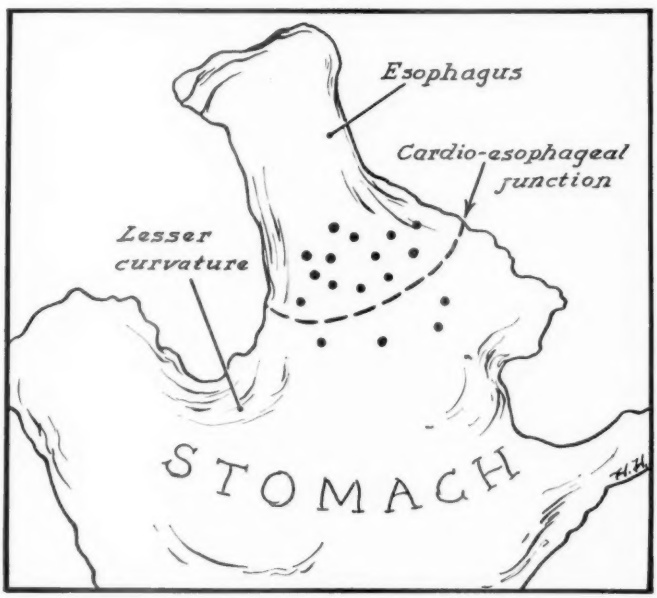

Fig. 1.—Graph showing sites of bleeding from single varix in 17 cases, 13 above the cardio-esophageal junction and 4 on the gastric side of the junction.

into the esophagus. At this time, following a suggestion of Dr. Sanford French, a rubber-shod gastrointestinal clamp is placed across the fundus of the stomach. This usually will stop the bleeding until the mucous membrane of the lower esophagus can be exposed. The clamp then is released if the bleeding point is not obvious. If found, stitch ligatures are placed above and below the varix, and the various wounds are closed in layers. If the patient is in satisfactory condition, portal pressures and liver biopsy are taken for the record.

As noted in Table II, a single bleeding point was found as the major offender in 17 cases. It is of interest that four of these patients bled from a gastric varix. In all 17 cases, the bleeding point was within 5 cm. of the gastro-esophageal junction (Fig. 1).

Biopsy of two of the three cases where only a generalized oozing was found was described as "esophagitis," but in no instance where a single bleeding point was found did there appear to be any gross evidence of inflammation.

TABLE II. SITES OF HEMORRHAGE IN OPERATED CASES

NUMBER OF CASES	23
Esophageal varix	13
Gastric varix	4
Duodenal ulcer	1
Generalized oozing	3
No source found	2

In Fig. 2, there is shown a bleeding varix at necropsy in a patient who died on induction of anesthesia from aspiration of gastric contents. The esophagus is pale and soft and the vein wall is thin. The findings in this series are in contradistinction to reports such as that of Chiles[4] who found ulceration of the esophageal mucosa at necropsy in 45 of 80 patients dying of hemorrhage.

Fig. 2.—Probe in bleeding point of esophageal varix. Necropsy specimen in patient who died after induction of anesthesia. Note absence of any evidence of inflammation in the esophagus.

In the second patient operated upon in this series in whom a bleeding point was found to be coming from a gastric varix, it appeared that geyserlike flow was pulsatile in nature. Furthermore, stitches placed in the varix above and below the bleeding point failed to stop the bleeding. The vein was incised and a small bleeding communication in the wall of the varix was noted. Samples of venous blood from the varix, from a peripheral vein, and from the artery were taken. There appeared to be a greater blood oxygen saturation in the varix. Since that original case, similar findings have been noted in three other cases. The oxygen data are noted in Table III.

It was presumed that the small arteriole in the wall of the varix represented an arteriovenous communication. Such large communications have

been described in the stomach wall.[1] In normal patients, injection studies of the arteries in the vicinity of the cardia show a marked variation in the extent of the arterial supply.[8]

It is conceivable that the level of the portal pressure may be influenced by these arterial anastomoses and that they may play a part in the difficulty in controlling hemorrhage. In fact, Taylor and Egbert[16] actually have suggested that esophageal varices may be congenital in origin and simply a variant of the vascular supply just as the arteries and arteriovenous communications vary.

TABLE III. OXYGEN SATURATIONS IN FOUR CASES OF BLEEDING VARIX

CASE NUMBER	ARTERIAL (PER CENT)	VARIX (PER CENT)	PERIPHERAL VEIN (PER CENT)
2	96	88	79
10	92	84	72
16	93	84	76
19	95	88	82

As a control group, similar oxygen determinations were made on 20 patients who had laparotomy performed for surgical lesions other than cirrhosis of the liver. The oxygen saturation of the portal venous blood was routinely approximately equal to that of the peripheral blood.

The operative mortality rate in these 23 patients was 32 per cent. All four patients who were jaundiced at the time of operation died. Two died from generalized bleeding from the wound and other mucous membrane surfaces in spite of rapid suture of a bleeding point. Two others died in cholemia within 48 hours. Two other patients died after control of hemorrhage in cholemia associated with delirium tremens. One patient died on the tenth day following leakage from the esophagus with empyema, followed by jaundice and cholemia. One patient died from massive aspiration into the trachea on induction of anesthesia. Following this experience, the intratracheal tube always has been placed first using local anesthesia, and the cuff inflated before the general anesthetic is begun.

TABLE IV. RESULTS IN 17 CIRRHOTIC PATIENTS SURVIVING OPERATION 3 TO 26 MONTHS

Died of cholemia	6
Died of hemorrhage	0
Living, but with further hemorrhages noted by fall in hematocrit or vomiting of small amount of blood	6
Living, no further bleeding	5

In four of the six cases in whom further bleeding was noted, a portacaval anastomosis was performed with no further bleeding in three of the four patients. In a fifth case, a portacaval anastomosis was attempted, but could not be performed. This patient has not bled again.

In three of the five cases in whom ligation only was performed, and no further bleeding occurred, a barium study of the esophagus shows that the varices have disappeared. This same sequence of events has been noted by Linder.[9] Linder's case was followed 16 months while our longest case has been followed by x-ray for only one year.

Finally, hepatic occluded pressure measurements were made in six patients with cirrhosis of the liver who were followed for varying periods of time. It is apparent from Table V that although none of these patients bled again massively, portal hypertension was still present to a degree approaching the level before the operation in all except one case.

TABLE V. HEPATIC OCCLUDED PRESSURE MEASUREMENTS FOLLOWING VARIOUS OPERATIVE PROCEDURES FOR BLEEDING IN CIRRHOTICS

PATIENT	OPERATION	PREOP. PRESSURE	POSTOP. PRESSURE	LENGTH OF FOLLOW-UP (MONTHS)	PRESSURE AT FOLLOW-UP
A. R.	PCA	350	260	49	330
F. L.	SRA	220	170	12	220
V. H.	PCA	420	206	25	300
H. B.	Lig. Var.	–	320	9	300
E. R.	Lig. Var.	–	360	12	310
J. G.	PCA	380	170	12	200

DISCUSSION

It is an interesting fact that practically every operative procedure which has been advocated as a method of therapy for bleeding esophageal varices has a small percentage of salvaged cases, just as in those cases where a local varix has been ligated. It certainly seems reasonable to believe that arrest of a massive hemorrhage must be lifesaving.

What happens afterward in those cirrhotic patients who survive any large intra-abdominal procedure is not so certain. It is difficult to understand why multiple varices should disappear after ligation of a single varix, as in Linder's case or as happened in three of our cases. It is equally difficult to understand why a few patients who have had the Talma operation,[18] or ligation of hepatic arteries, or any of the other operations reported should have their successes, unless we are willing to believe that the trauma of the operation itself has created an extensive collateral which did not exist before the operation. Sherlock, who has studied more than 100 splenoportagrams in cirrhotic patients, has shown on the lateral x-rays that the retroperitoneal collateral in the cirrhotic patient is enormous and of a considerably greater diameter than the 7 to 10 mm. anastomosis constructed by operation. And yet, the advocates of portacaval anastomosis have ascribed credit for salvage to the procedure itself.

It is a well-known fact that the estimated hepatic blood flow is decreased in the cirrhotic patient.[2] It also is a well-known fact that the circulating blood volume is increased in the cirrhotic patient.[12] The assumption that an extensive retroperitoneal collateral must be present is the only way one can account for the ability of the increased volume of blood to get back to the heart. If one agrees with Ogilvie,[13] "the surgeon divides a number of natural anastomoses whose total cross section is much greater than the rather poor one he is about to construct," one must wonder whether the effect of the operation is a specific one.

When one adds to this uncertainty the evidence of hepatic occluded pressure measurements, which would suggest that the portal hypertension recurs

after a variable period of time, and the reported cases of fatal hemorrhage in cirrhotic patients in the presence of measured normal portal pressure, it would seem reasonable that those few cirrhotic patients who survive any surgical procedure are the ones with the best prognosis anyway and that if the immediate crisis can be weathered, they will live the same length of time irrespective of the therapeutic procedure. Therefore, we feel that the simplest and most direct attack upon the bleeding point is the best operation for the bleeding cirrhotic patient and that any sort of prophylactic procedure is not justified.

SUMMARY

1. Twenty-three consecutive patients with massive gastrointestinal hemorrhage thought to be due to esophageal varices are presented.

2. The difficulties encountered in attempting to find and ligate a bleeding point are discussed.

3. Ligation of a bleeding varix during acute hemorrhage can salvage a few cirrhotic patients for an additional period of time.

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DISCUSSION

DR. JULIAN JOHNSON (Philadelphia, Pa.).—Dr. Linton has talked about it a good many times, and I hope he will say something about it here today.

There is one point I would like to make. It seems to me there is a place for a so-called "semi-emergency" for caval shunts in this type of patient. I do not think there is much point in operating upon the patient who is unconscious with jaundice and that sort of thing.

The type of patient I am referring to is one known to have cirrhosis with esophageal varices, is in relatively good shape, and has been under observation in our clinic, when he suddenly comes in with gastrointestinal hemorrhage. The moment he comes into the hospital, we put down the Blakemore tube to stop his hemorrhage. It seems to me that that patient, if he has not lost too much blood, is apt to be as physically sound then as he would be at any future time. We prefer to watch such a patient for a day or two and evacuate any residual blood from his bowel. If he seems to be recovering satisfactorily and has not required too much blood to replenish his loss, I believe it is justifiable to perform a portacaval shunt at that time. The Blakemore tube is usually left down for an additional 4 or 5 days. We have not had cause to regret this type of maneuver. We have performed about six of them.

Two of them, I suppose, we should not have done. One of them was slightly jaundiced, having bled once, and came in our ward and waited. We thought we had the bleeding stopped but it bled again. She survived the operation in spite of jaundice. The only one we lost, we were too enthusiastic about. He was in a semicoma at the time we operated and doubtless should not have been operated upon at that time. We have hesitated to subject these apparently good-risk patients to ligation of the varicose veins as an emergency procedure for fear that they would bleed again before they recover sufficiently for the portacaval shunt. We have had this occur in 2 patients.

To do the portacaval shunt is not a time-consuming or difficult operation in many of these patients. We like to turn them with the right side up, but start out with the abdominal part of the incision. If there is any doubt as to the diagnosis, a catheter may be placed in a mesenteric vein and pressures recorded. A portagram may also be made to be sure the portal vein is adequate. In the average person, we can perform the procedure in 2 to 4 hours. This seems to us to be a justifiable method of handling certain carefully selected patients.

DR. ROBERT R. LINTON (Brookline, Mass.).—I would like to take this opportunity to thank Dr. Cohn for presenting a very excellent discussion of this interesting problem of bleeding esophageal varices. In many clinics I still think that the subject has not been clarified as well as it has in our hospital. Many surgeons have become discouraged with splenorenal shunts and have tended to resort to the direct portacaval shunts because of their greater ease of construction. I believe, however, as the long-term results are tabulated, we will find that those surgeons who are able to construct a satisfactory splenorenal shunt will show a higher percentage of good results.

I disagree with Dr. Julian Johnson in that, in my opinion, it is extremely rare that an emergency portacaval shunt should be done for bleeding esophageal varices, because in most instances, one will be performing a major operative procedure on an extremely ill patient without a proper evaluation of the patient's general condition—and especially that of a damaged liver in many of them. Furthermore, to expedite the operative procedure, one is almost limited to performing a direct portacaval shunt which, in our hands, has resulted in a higher mortality rate than a splenorenal shunt when dealing with patients with seriously diseased livers. For some reason we have not been completely able to fathom, something happens to these livers when you interrupt the portal vein to do a direct portacaval anastomosis which does not occur when one does a splenorenal type of shunt.

In my own experience, in one year I lost 5 patients following direct portacaval shunts. Two occurred immediately postoperative, and three within a year from the time the shunts were performed, and all of them from liver failure. It is my opinion, therefore, that the suturing of the esophageal varices, as a first stage, in patients whom it is necessary to balloon tamponage to control their esophageal bleeding and then 6 weeks later performing a splenorenal anastomosis in a majority of them is the better method of handling these serious problems.

I agree with Dr. Cohn in his method of controlling the esophageal bleeding by suturing the varices, but personally I prefer to do the operation through the chest since it can be performed with less depth of anesthesia and by opening the stomach, as well as the esophagus; at the cardioesophageal junction one can suture the large gastric varices in the upper part

of the fundus as well as those in the lower part of the esophagus. Another advantage of performing the operation through the chest is that it leaves the abdominal cavity free for the shunt procedure.

DR. ARTHUR H. BLAKEMORE (New York, N. Y.).—I am unalterably opposed to a plan of emergency management (traction tamponade of the stomach employing a single gastric balloon) that commits the surgeon routinely to the necessity of performing an emergency operation for the control of bleeding from esophageal varices in patients with cirrhosis of the liver.

The high percentage of patients with cirrhosis that have badly damaged livers plus the rapid deterioration of the liver that follows the onset of hemorrhage establishes an unalterable pattern of a high mortality rate following emergency surgery, whether it be suture of varices or the so-called emergency portacaval shunt. A sample of the mortality rates with these respective emergency procedures has been presented at this meeting. In consideration of the above facts, we take the view that emergency surgery should only be employed in those patients with ruptured varices of the stomach wall. Bleeding from varices of the stomach wall can only be arrested by traction tamponade of the stomach—a method which, because of poor patient tolerance, deadlines to emergency surgery. Fortunately, emergency surgery can be avoided in patients with bleeding varices of the esophagus by employing correctly a properly designed esophageal balloon. This is important because bleeding from varices of the esophagus accounts for an estimated 90 per cent of all cases of cirrhosis with bleeding varices.

The adult human esophagus measures approximately 3 cm. in diameter. The range of physiologic distention of the upper half of the esophagus is minimal, whereas the lower half of the esophagus ranges up to 6 cm. in diameter. With the above facts in mind, the sausage-shaped esophageal balloon to function properly in the control of bleeding from a ruptured varix in the esophagus must be at least 3 cm. in diameter and the wall must be sufficiently thin to dilate the esophagus to physiologic limits (6 cm. lower half) at minimal manometric pressure. With this accomplished, continued inflation of the balloon to a pressure (45 mm. Hg), slightly higher than the highest pressure recorded (44 mm. Hg) by direct measurement in cases of portal hypertension of necessity causes collapse of the esophageal varices with cessation of hemorrhage.

The above hypothesis was completely confirmed over nearly a 2-year experience with handmade, double-balloon nasogastric tubes employing prophylactic rubbers of suitable length, diameter (3.5 cm.) and wall thickness (.0006 in.). The patients showed good tolerance for esophageal balloon tamponade over protracted periods. With the result that patients with evidence of severe damage to the liver such as deep jaundice, central nervous system symptoms of impending coma and, in a few instances, frank coma were safely shepherded through a bleeding attack—patients in whom there would be little likelihood of survival had they been subjected to emergency surgery.

Unfortunately, with the manufacture and marketing of the nasogastric double-balloon tube for diagnosis of the bleeding site and differential tamponade, there has, on occasion, occurred deviation from the afore-mentioned specifications. The most serious deviation to occur in manufacturing the tube was increasing the wall thickness of the esophageal balloon to the point of an ineffective degree of expansion when inflated to a given pressure. The manufacturer is in the process of correcting the fault in the product and anticipates replacement of the existing tubes when the standardized product is in production.

DR. ROY COHN (closing).—Part of the reason for our study was that we wanted to follow patients who had had ligations only, to see if they did as well as those who had portacaval shunts performed. Roentgenograms on patients 6 months after a single ligation showed that the varices had disappeared. Fortunately, all these cases are in jail so we will have a good chance to follow them.

The other point is that Sherlock, working at Hammersmith, has shown by percutaneous splenoportagrams that there is, in some cases, an enormous amount of retroperitoneal collateral and it is hard to believe that the anastomosis of veins 1.5 cm. in diameter at the most will actually lower the pressure to the extent which has been set. I think the British generally feel that way; but, as has been mentioned, none of these problems is settled by any manner of means.

SUBTOTAL PERICARDECTOMY FOR CONSTRICTIVE PERICARDITIS

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THE operation "pericardectomy" is one of the oldest surgical procedures upon the heart. The history of this operation, when traced up to the present, is characterized by an increasingly radical approach to the problem. Wider areas of the affected pericardium are being excised through ever-lengthening incisions. The early approach was through a left anterior incision, but soon these were extended to include resection of costal cartilages and extension into the sternum. Emile Holman,[6] who has done much to draw our attention to the necessity of a radical operation, has advocated a sternal splitting incision, while recently, Johnson and Kirby[7] and the authors have suggested the use of a transternal approach.

As one studies the results, there does seem to be some correlation between better results and a more radical excision. It is not possible from the literature to draw any such conclusions, but the authors suggest that it is a significant enough impression to warrant careful study.

This is a preliminary report of a series of 13 cases that underwent a radical excision of the constricting pericardium or a subtotal pericardectomy. The longest follow-up is only 3 years, and an attempt has been made to determine whether this radical excision will eliminate postoperative cardiac failure and late recurrence of symptoms after operation, as well as allow a return to normal or near normal hemodynamics. In reports in the literature, many of the deaths, failures, and late recurrences have been blamed on myocardial scarring or myocardial atrophy with failure. Only a careful follow-up will determine how important this factor is. Perhaps this only plays a minor role and the important cause of failure is inadequate excision.

IMPORTANCE OF RADICAL EXCISION

Pathology.—Some of the early writers and most of the recent writers (Schmeiden,[9] Holman and Willett,[6] Heuer and Stewart,[5] and Harrington[4]) agree that the constricting pericardium should be excised extensively, including the apex and the diaphragmatic surface of the ventricular chambers. Very few, if any, appear to remove the pericardium posterior to the phrenic nerve. This may be an important omission.

Read at tenth annual meeting of the Society for Vascular Surgery, Chicago, Ill., June 10 1956.

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There is general agreement that removal of the pericardium from the right and left atrium is not overly important and is fraught with a danger of entering the cardiac chamber.

There is no real clinical evidence that constriction of the venae cavae at the time of operation is a cause of signs or symptoms of constrictive pericarditis. Blalock and Burwell[3] and Sellors[8] state that freeing the venae cavae is not necessary. From the operative description of other writers, it would appear that the venae cavae have not been freed. Beck[1] and Harrington[4] state that they should be freed but this would appear to be based on a clinical impression. Holman and Willett[6] advocate freeing of the venae cavae and have reproduced the signs of this disease experimentally by partially obstructing the venae cavae in animals.

There is very little mention of the value of freeing the pulmonary vein or the root of the lung.

Failure of Operation.—Many of the early reports in the literature cite an incidence of no relief or incomplete relief of signs and symptoms. Some cases showed a recurrence after initial relief. Paul White and associates[10] and others[5, 6] have reported cases in which a second operation with more extensive resection of the pericardium was necessary with resulting improvement. The majority of these initial operations were carried out through a left-sided incision.

TABLE I. PERICARDECTOMY
(Toronto General Hospital 1939-1950)

TOTAL CASES	POSTOPERATIVE DEATHS	RECURRENCES	ALIVE AND WELL
10	4	2 (1 and 3 years)	4

The results of pericardectomy performed at the Toronto General Hospital between 1939 and 1950 are typical of these early reports (Table I). Ten pericardectomy operations performed by 3 surgeons showed an operative mortality in 4 cases, 1 in 5 hours and 3 on the second and third postoperative day. These deaths were all considered cardiac in origin. A unilateral incision was used in each case. Of the 6 survivors, 2 patients showed improvement and returned to light work. After initial improvement, 1 of the 2 developed a recurrence of symptoms a year later and died at the age of 31. The second developed recurrence of signs and symptoms 13 years later and died at the age of 48. Four of these patients are still alive and well.

It is not possible to compare an early and late series, for one must consider improved methods of anesthesia, supportive therapy, and antibiotic therapy. However, recent reports of operations with wider excision of the pericardium have shown a reduction in the percentage of failures. It remains to be seen if an even more radical approach will further reduce or eliminate failures and what part myocardial atrophy plays in the problem.

SUBTOTAL PERICARDECTOMIES

Indications.—It would appear that all cases that have been diagnosed as constrictive pericarditis should be operated upon unless there are special

complicating factors which would contraindicate surgery. As for early diagnosis, it appears that a case seldom presents itself prior to the development of increased venous pressure and even ascites. One problem which may present itself is the case which is found on routine x-ray examination of the chest or in a sanitarium.

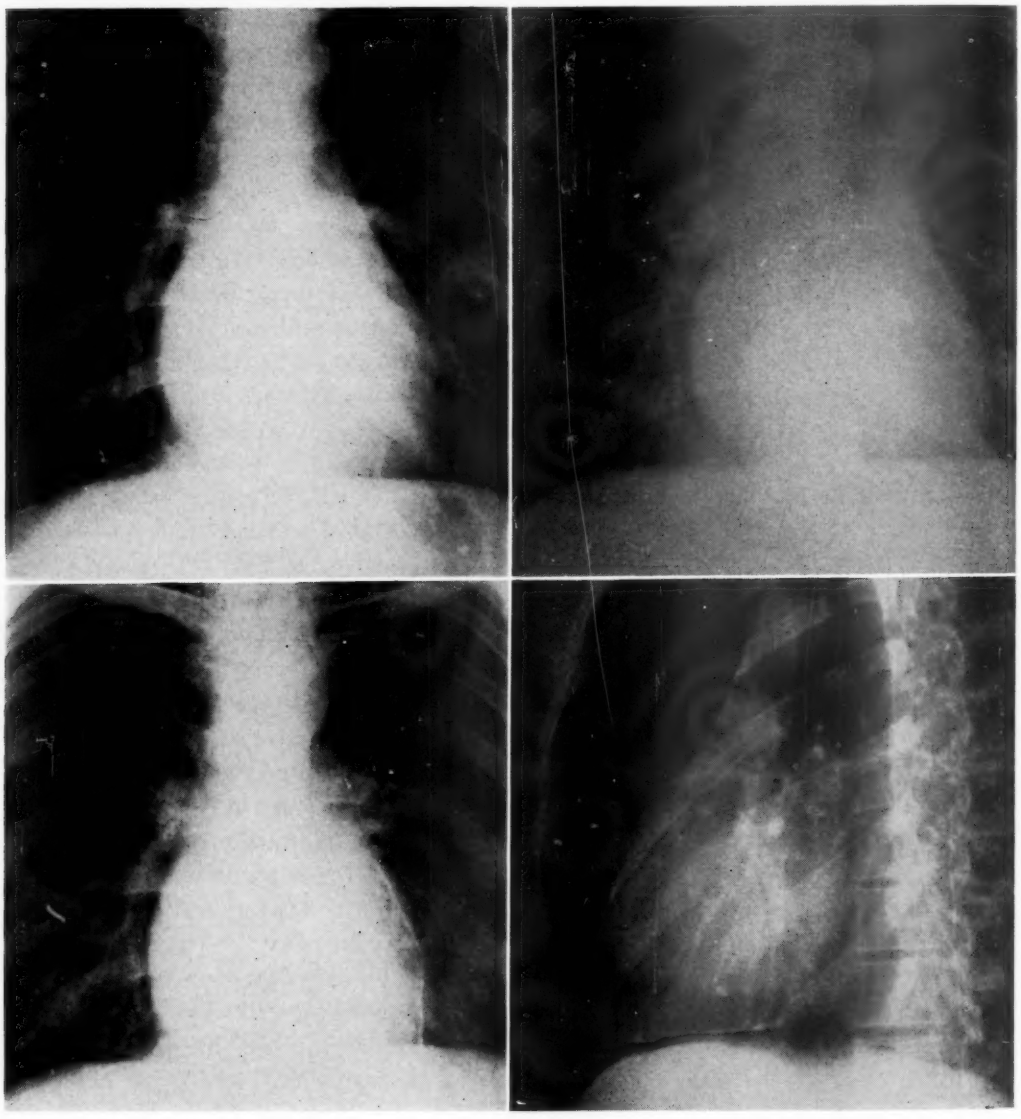

Fig. 1.—These are some radiographs of the same patient. They show the rate at which calcification may occur in the pericardium. *A*, 3½ years before operation, *B*, 2½ years before operation, and *C* and *D* at time of surgery.

One such case is included in this series. He was a soldier with apparently normal exercise tolerance. A chest plate prior to discharge from the army showed advanced calcification which was so minimal two and a half

years previously that it had been overlooked. Fig. 1 demonstrates the radiologic changes in the patient, and it illustrates the surprising rate at which this process of calcification can spread. Examination showed a moderate increase in venous pressure and moderate restriction of heart movement on fluoroscopic examination. The findings on cardiac catheterization showed high end-diastolic right ventricular pressure and the classical pressure tracing found in constrictive pericarditis. In spite of the lack of serious symptoms, these findings along with extensive calcification were considered enough evidence to warrant surgery.

Two of the 13 cases to be reported had an active tuberculous process three months previously and were still on active tuberculosis therapy. Both patients were in chronic failure and had recently ceased to respond adequately to diuretics. Pericardectomy was carried out as an urgent procedure in each case with satisfactory results. The tuberculosis therapy was continued and they were transferred within a few weeks to sanitarium.

Preparation.—All 13 patients were placed on the usual strict cardiac regimen preoperatively with digitalis, diet, and diuretics until their condition was considered optimum and their weight steady and minimal. One early patient was operated upon without digitalis therapy. He developed some dilatation of his heart after removal of the pericardium and did very poorly for two days after operation until he was digitalized. Coincident with this, his improvement was dramatic. One other patient came to surgery without digitalis, and when the pericardium was removed his heart showed rather marked dilatation. Intravenous digitalis therapy on the operating table showed an increase in muscle tone and a reduction in heart size which appeared to coincide with the effect of the digitalis. With these two early cases in mind, all patients have been digitalized preoperatively and remained on digitalis for a varying period after operation until it was felt they had obtained the optimum improvement.

Four patients with marked pleural effusion and ascites unrelieved by medical treatment required aspiration the day before surgery as an aid to proper anesthesia.

Technique.—It is the feeling of the authors that none of the unilateral incisions, even though they involve the removal of costal cartilages, will permit the surgeon to carry out a safe radical excision of the pericardium. The venae cavae and region of the atrioventricular groove are not readily accessible without manual retraction and compression of the heart which is tolerated poorly. To properly manage accidental tears of the heart wall may require better exposure than these unilateral incisions will give.

It is our opinion that the transternal incision allows one to carry out the wide excision or subtotal pericardectomy with the greatest safety. There is no special advantage in the midsternal incision from the point of view of avoiding entry into the pleural cavity. It is our impression that it would be impossible to adequately resect the pericardium without entering both pleural cavities. The important thing to remember in a transternal incision is that opening or spreading the wound occurs principally by virtue of the upward movement of the superior segment. The inferior wound edge is relatively stationary. Thus the incision should be placed over the lowest area to be exposed. The actual

position will vary with heart position, but the fourth space on the right and fifth space on the left have been found satisfactory in most cases. A V-shaped cut is made across the sternum to aid in accurate realignment of the wound. The sternum has solidly united in all cases. The pericardium overlying the anterior and diaphragmatic surface of the ventricle is excised together with the left lateral surface of the heart after freeing and retracting the phrenic nerve. On the left side, the tip of the left auricle is usually exposed and the left pulmonary vein freed. Usual care is taken in the region of the anterior descending branch of the left coronary and the atrioventricular groove. Dissection is usually not carried up on the great vessels but is extended to free the superior and inferior venae cavae. The removal of the pericardium from the body of the right atrium is only carried out if this can be done with ease. The left pulmonary vein is also freed in the region of the lung root.

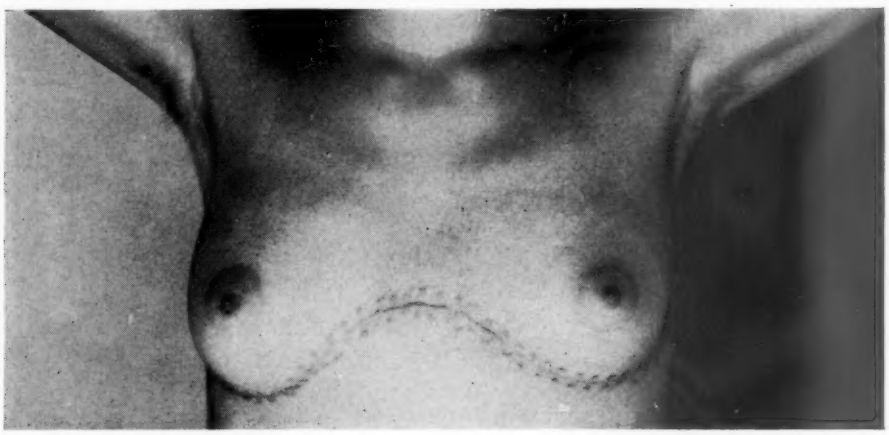

Fig. 2.—The transternal incision in a female patient. Usually the fourth interspace on the right and the fifth space on the left side is entered.

It has been found in most cases that there is a circular area surrounding the apex of the heart that is not involved in calcification. It is the mobility of this apical area that proves misleading on fluoroscopy. Dissection is started in this area and the left ventricle is freed first. The chest is closed with closed drainage on each side, and no serious attempt to reconstruct the mediastinal pleura is made. The important thing in closure is to use fairly heavy wire sutures. Harris wire tyers are employed to hold the sternal bone fragments very firmly and somewhat impacted. This is necessary if one is to avoid postoperative wound pain. Fig. 2 illustrates the incision in a woman, and Fig. 3 indicates the very adequate exposure obtained by this approach.

Hypothermia.—Four of the 13 cases were operated upon under hypothermia at a body temperature of 30° to 31° C. They were considered very poor-risk patients. Hypothermia is not used as an adjunct in poor risk cases with any thought that it specifically combats shock. The principle may be simply stated.[2] Hypothermia affords some protection to the heart from the deleterious effects of hypoxia. It should be used in an operation where, due to manipulation of heart and lungs, one is likely to cause periods of myocardial hypoxia and when a patient tolerates hypoxia poorly. With proper control of blood pH during

cooling and rewarming and at a body temperature not lower than 30° C., this is a relatively safe procedure. It is the authors' opinion that this reduces the mortality rate in very poor-risk cardiac surgery.

ASSESSMENT OF CASES AND RESULTS

Table II gives a summary of the assessment of the 13 cases presented for study. The average age was 29 with a variation from 16 to 56 years of age. Twelve of the 13 showed signs of right heart failure with an average duration of 8½ months. The venous pressure in all patients was elevated with an average pressure of 24 cm. of saline.

Fig. 3.—A view of the operative field with a transternal incision. At the completion of the operation, the pericardium will have been resected as completely as possible, including the freeing of the venae cavae, lung roots, diaphragmatic surface, and the area posterior to the phrenic nerve. This provides excellent exposure and good control of any accidental injury to the myocardium.

TABLE II. CHRONIC CONSTRICTIVE PERICARDITIS

Number of cases	13
Average age	29
Signs of right heart failure	12
Average duration of signs	8½ months
Average venous pressure	24

The disability varied in these patients, but many of them were very advanced and debilitated with massive pleural effusion and ascites. One patient had been completely bedridden for one and a half years with bilateral effusion and ascites with cyanosis and cachexia. The two patients with acute tuberculous processes have already been referred to.

Table III indicates the results of subtotal pericardectomy upon this group of 13 patients. There was one operative and one postoperative death, and on

the basis of a follow-up of 1 to 3 years, there has been no recurrence of symptoms in the 11 survivors.

TABLE III. SUBTOTAL PERICARDECTOMY

TOTAL CASES	DEATHS		RECURRENCES 1 TO 3 YEARS	MORTALITY RATE (PER CENT)
	OPERATIVE	POSTOPERATIVE		
13	1	1	0	15

The operative death was due to pre-existing, unrecognized, coronary disease. At autopsy the circumflex branches of both the right and left coronary arteries were found to be obstructed by calcification surrounding the vessel, and the terminal branches of the remaining anterior descending branch of the left coronary were obstructed by fresh thrombus sufficient to produce myocardial ischemia.

Cardiac massage was carried out on this man for three and one quarter hours during which time, with reduction of anesthetic, the patient would awaken and respond very intelligently to all questions. It became obvious that the coronary circulation for some reason was inadequate and resuscitation was stopped.

The second death was that of a man aged 33 who had been denied operation because of his advanced pulmonary fibrosis. He had eventually developed marked cardiac cirrhosis with evidence of serious damage to his liver. The patient was in a terminal state. The operation was carried out very satisfactorily and the patient's immediate postoperative course was excellent. He was sitting up and quite strong on his third postoperative day. Following this, he developed evidence of hepatic failure and died in acute hepatic coma with necrosis of the liver found at post mortem.

Of the 11 survivors, one was operated upon too recently for proper assessment. The remaining 10 represent a follow-up from 14 months to 3 years with an average follow-up of 2 years. They have been assessed as 7 excellent, 2 very good, and 1 fair result. The latter patient is gradually improving. He represented a very terminal stage of the disease at operation. If his cardiac function does not eventually come to within normal limits, one may have to consider this due to irreparably damaged myocardium. (See Table IV.)

TABLE IV. RESULTS OF SUBTOTAL PERICARDECTOMY
(Follow-up 14 months to 3 years)

CONDITION	NUMBER OF PATIENTS
Excellent	7
Very good	2
Fair	1 (improving)
Total	10

SPECIAL STUDIES

The reader may be reminded that this is a preliminary report, and no conclusions may be drawn from this number of cases. However, it was felt worth while to indicate the special studies which are in progress and the results obtained thus far.

Fig. 4 illustrates the venous pressure, circulation time, total blood volume, and cardiac output studied preoperatively, 4 to 6 weeks postoperatively,

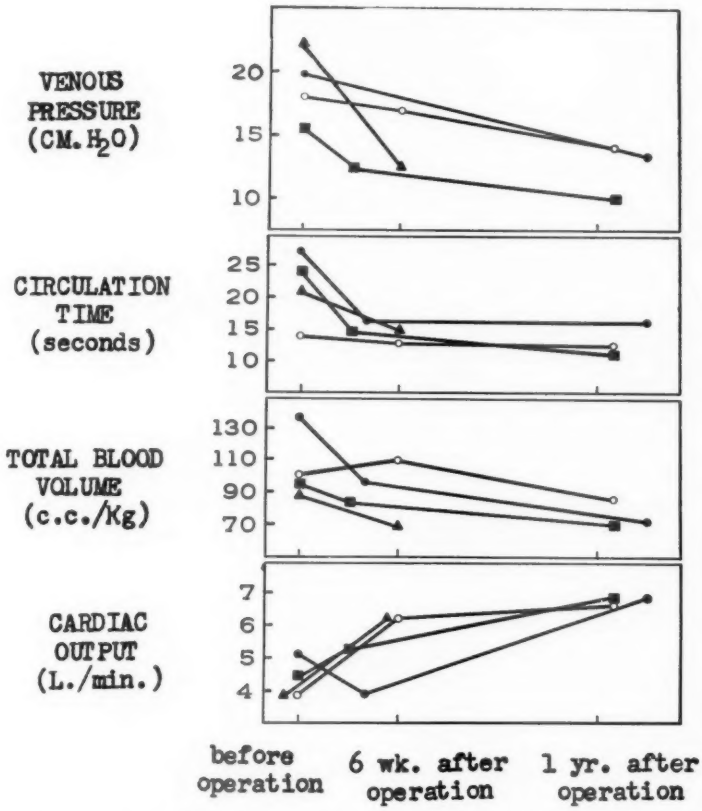

Fig. 4.—A graphic illustration of special studies in progress before and after subtotal pericardectomy. It is interesting that in the 3 patients studied one year after operation there has been evidence of continued improvement in cardiac function.

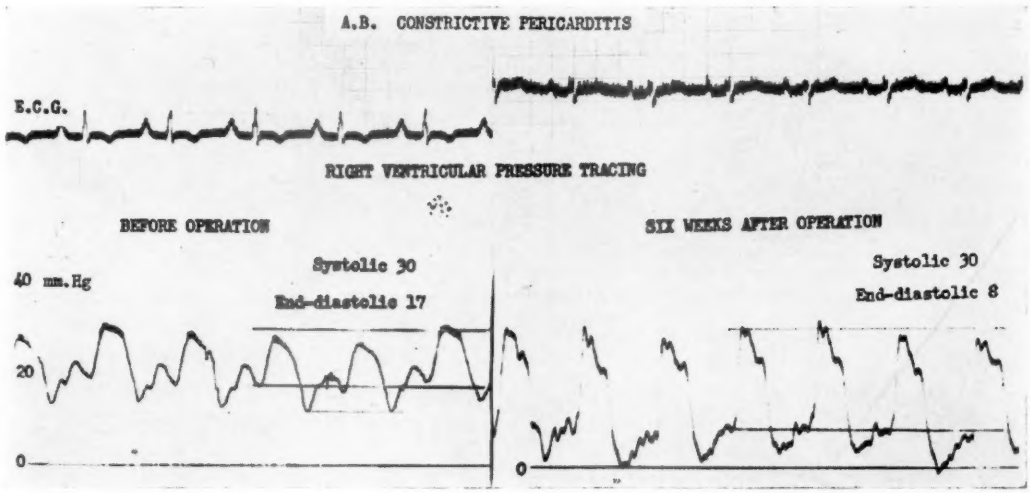

Fig. 5.—Typical right ventricular pressure tracings before and after operation. Preoperatively the "end diastolic pressure" was 57 per cent of the systolic pressure. After operation this ratio was well under the normal maximum of 30 per cent.

and approximately 1 year postoperatively on 4 patients. There is an improvement in all four estimations immediately after operation. The interesting thing is that they all appear to continue this improvement during the ensuing year which would suggest improvement in myocardial function approaching normal.

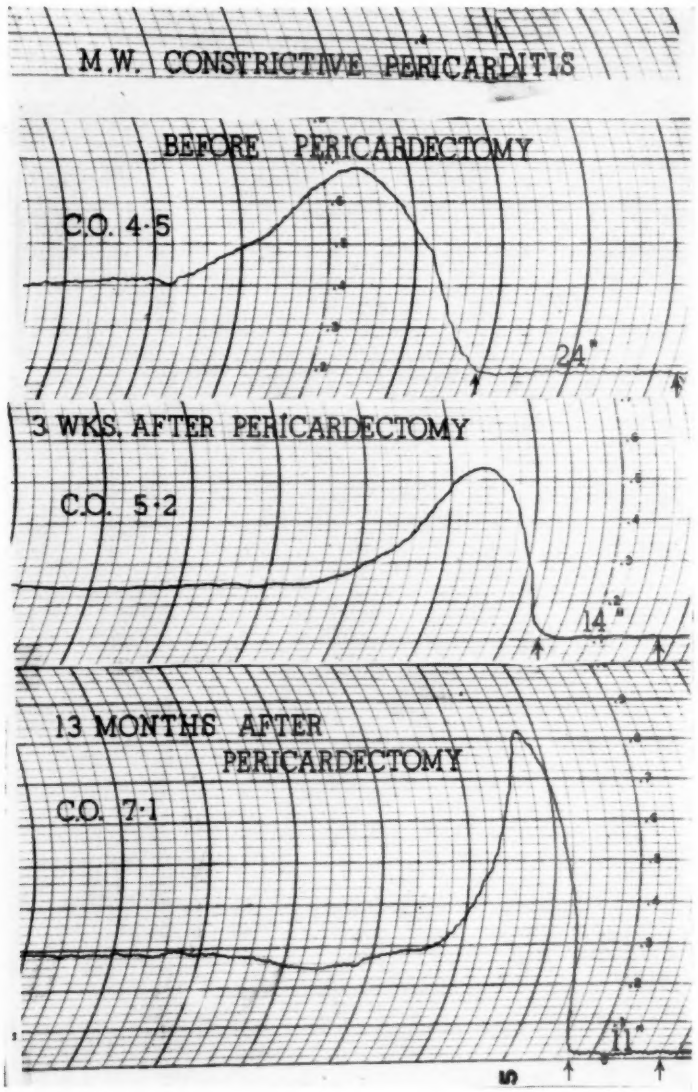

Fig. 6.—Illustration of the curve obtained using the Hamilton dye dilution principle. This illustrates the return to normal over a period of 13 months of the cardiac output circulation time and shape of the curve.

Fig. 5 indicates the typical right ventricular pressure tracings seen on cardiac catheterization before operation and after operation. Normally, the end diastolic pressure should not be more than 30 per cent of the systolic pressure. In the preoperative reading, the end diastolic pressure was 57 per cent of the systolic pressure which is considered typical in constrictive pericarditis. In the postoperative tracing, this high end diastolic pressure has disappeared, and the relationship of diastolic to systolic pressure is now within normal limits.

The circulation time, total blood volume, and cardiac output have been calculated by one of us (Gunton) on the Hamilton dye dilution principle. Fig. 6 illustrates the dye dilution curve obtained preoperatively, 3 weeks, and 13 months after operation. This illustrates graphically the return to normal over a period of time.

SUMMARY

1. The importance of a radical pericardectomy for constrictive pericarditis is discussed.

2. Operative failures and recurrences may be due to myocardial atrophy or inadequate excision of pericardium.

3. A preliminary report of 13 subtotal pericardectomies is presented with results based on a 1- to 3-year follow-up.

4. Clinical results and hemodynamic studies thus far would suggest that an important factor in success is adequate excision of pericardium.

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DISCUSSION

DR. FRANK GERBODE (San Francisco, Calif.).—I could not let this moment go by without saying a word about this very interesting and recurring problem of the surgical treatment of constrictive pericarditis, particularly as Dr. Holman from our clinic has been so interested in it and has advocated earlier operation in tubercular pericarditis. I wish to compliment Dr. Bigelow on his usual ability and interest in studying the physiologic responses of the patients being operated upon, and also for his desire to increase the thoroughness of the operation.

I am sure the transverse incision which he advocates is an excellent one and, although we have employed the sternal splitting incision in many of our cases, I have found that the transverse incision frequently gives a better exposure.

I would like to comment one moment on hypothermia in these cases. We debated on whether it was advisable to use hypothermia in this group of patients and others who have serious heart disease and decided that although it might have some value there were obvious disadvantages to its use. We have therefore kept these patients and most of those who were being operated upon for mitral stenosis on 100 per cent oxygen during the cardiac portions of the operation. In about 200 operations it has been possible to maintain the patient on pure oxygen for about an hour and a half. It has been apparent that this results in a more stable heart which remains pink and stands intracardiac procedures better than when an inhalation anesthetic is given.

THE ATYPICAL DUCTUS

Physiologic and Technical Considerations in Its Surgical Therapy

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WITHIN the large group of patients with persisting patency of the ductus arteriosus, there is a comparatively small number who have what has been designated as the 'atypical ductus.' This group, which comprises 6 to 18 per cent of all cases,[7, 12] is important since both diagnosis and therapy are quite different from the usual patient with a patent ductus. The two principal features which distinguish the 'atypical ductus' are its large diameter and the presence of hypertension in the pulmonary circulation. Ten cases will be used to illustrate the consequences of this altered anatomy and physiology as they affect the practical considerations of surgical treatment.

Alterations in the pulmonary blood vessels which accompany long-standing hypertension have long been recognized. Recently many reports have discussed the etiology of these changes, especially in association with patent ductus arteriosus.[8, 11, 18, 26, 30, 31] For the purposes of this paper it is sufficient to recognize that pulmonary hypertension is associated with the presence of thickening in the walls of the pulmonary arteries and that this thickening, when it involves the smaller vessels, introduces an abnormal resistance to blood flow through them. Edwards[11] has pointed out that these vessel walls resemble those found in fetal lung and suggests that normal postnatal thinning may be prevented by the excessive flow accompanying persistent patency of the ductus arteriosus. He also believes that changes so initiated are progressive and irreversible, even when flow through the lung is reduced by the increasing vascular resistance. Whether they are also irreversible if excessive flow is eliminated by correction of the underlying anomaly is not clear. It is evident, however, that these changes are found in association with a ductus arteriosus only when its lumen is unusually large. When the diameter of the ductus remains nearly as large after birth as that of the aorta, the damping effect of a small-caliber, high-pressure ductus stream entering a large-caliber, low-pressure pulmonary arterial stream is diminished or lost, and the pulmonary vascular bed must accommodate not only an excessive flow, but also a flow at systemic pressure.

Physiologic compensation to excessive flow and pressure of this degree results in a syndrome which differs from that associated with the much more common smaller persistent patencies of the ductus. Its manifestations vary not only with the anatomic abnormality, but also with the stage in evolution

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of the compensatory mechanism which the patient has reached. He may present himself at any point in this evolution. At an early stage with a large left-to-right shunt, signs may differ little from the usual ductus. Unusually large heart size, a murmur which lacks the usual continuous quality and may be confined to systole, and more than ordinary nutritional or developmental handicaps are clues to presence of real hypertension. When the syndrome is more fully developed, right ventricular enlargement becomes dominant as the pressure rises in response to increasing pulmonary vascular resistance. Ultimately pulmonary hypertension advances to the point at which pressures at each end of the ductus are nearly equal. Now flow through the ductus diminishes. At this stage it is balanced in a precarious equilibrium with small volumes of blood passing through the ductus in both directions at different phases of the cardiac cycle. In the last phase of the syndrome, pulmonary resistance is greater than peripheral and reversal of ductus flow is complete.

Difficulties of a physiologic nature seldom arise following ductus closure in those patients with a moderate pulmonary hypertension. However, in the presence of severe pulmonary hypertension certain undesirable changes in cardiodynamics may appear when the shunt is closed. These are manifested by a further rise in the pulmonary artery pressures, dilatation of the right ventricle, and weakening of cardiac action, with prompt remission when the ductus is reopened. In such cases the flow through the ductus is large, and the total circulating blood volume is also much increased. Abrupt separation of the circulations may result in acute failure even in the absence of advanced pulmonary vascular changes. Failure may also occur at a later stage through a different mechanism. With advanced pulmonary vascular changes and balanced or reversed shunt, flow and total blood volume are no longer excessive. Now the ductus acts as an escape vent for the right ventricle which may not be able to empty through the narrowed pulmonary vascular bed. It becomes evident, therefore, that physiologic difficulties which accompany closure of the ductus will parallel the degree of pulmonary hypertension and the stage of secondary vascular change in the pulmonary circulation. In addition certain technical difficulties add to the hazards of ductus closure in this group. For this reason it seems worth while to present our experience.

From a group of 150 surgical closures of the ductus, 10 patients have been selected which show in varying degree the 'atypical' features which have been discussed. These are presented in order of increasing pulmonary hypertension. Although actual pressure readings were not obtained in all cases, the anatomic findings and physiologic response to closure indicate that they belong in this group and may be used to illustrate important features in management of the syndrome.

CASE REPORTS

CASE 1.—M. A. Shortly after birth a heart murmur was noted. Attacks of cyanosis with crying requiring oxygen occurred in the neonatal period. He was first admitted at 4 months for evaluation. At this time he was slightly cyanotic, and his heart was much enlarged with a systolic murmur over the precordium. Electrocardiogram showed complete block with nodal rhythm. On digitalis he did fairly well for 8 months, when he was readmitted because of respiratory infection and failure. With antibiotics and oxygen therapy he improved. Angiocardiogram showed prompt filling of a large right ventricle and dilated

pulmonary artery. Simultaneously the descending thoracic and abdominal aorta were opacified, but not the ascending aorta. Five seconds later the left heart and ascending aorta were opacified and the pulmonary radicles again filled. A diagnosis of patent ductus arteriosus with probable infantile coaretation and possible atrial septal defect was made.

A momentary cardiac standstill occurred immediately following the angiocardiogram, but the heartbeat returned promptly without massage. Thereafter ectopic ventricular foci appeared in the electrocardiogram. Two mild convulsive episodes occurred during recovery. For the next 6 months he continued to show transient cyanosis and remained slow in weight gain and development. It was decided that in spite of the known risk that closure of the ductus was indicated.

On admission at 18 months he showed systolic and diastolic murmurs in the third left interspace, palpable femoral pulses, complete heart block with evidence of atrial hypertrophy by electrocardiogram. Blood pressure was 100/40. Toenail beds were duskier than fingernail beds; the difference was accentuated on crying.

At operation a ductus 15 mm. in diameter lying in fetal relationship to an aorta of the same diameter was found. There was no coaretation. No thrill could be felt over the heart. The ductus was closed by mobilizing the aorta and producing gradual occlusion with a Potts-Smith-Gibson clamp, after which it was divided between this and a Potts ductus clamp and the cut ends were sutured. Blood pressure fell to 80/30 on release of the clamp, but rose within a few minutes to 120/80.

Postoperatively his color appeared improved but 4 hours later his temperature rose to 107° F., falling promptly with alcohol sponge. During the next 3 days he had several episodes of cyanosis convulsions and fever spikes to 104° F. In the intervals his color appeared good. During the following 4 weeks he had no further convulsions and appeared to be making a good recovery. One month later, 2 months after operation, while apparently in good health he suddenly had an episode of cyanosis and lost consciousness. He was immediately readmitted. During the next 8 hours he had 10 episodes characterized by sudden cyanosis followed by a convulsion and then apnea and apparent cardiac standstill for increasing periods. Electrocardiogram taken during these showed ventricular tachycardia. Ten hours after admission a more severe episode of the same character occurred and he expired.

At autopsy a high interventricular septal defect was present. The ductus closure was well healed. Definite arterial wall thickening was present in the smaller pulmonary arteries, characteristic of pulmonary hypertensive changes.

CASE 2.—S. G. A heart murmur had been noted at 1 week. At 7 weeks the murmur was continuous and there was marked right axis deviation on electrocardiogram. The heart was much enlarged and overactive. There was no cyanosis. She grew and developed slowly, having frequent respiratory infections with prolonged cough after each. At 6 months the continuous murmur was again noted, with accompanying thrill. Blood pressure was 150/20.

On admission at 21 months the symptoms and signs were even more marked. She weighed 20 pounds and was 31 inches tall. Respiratory difficulty was almost constant but there was still no cyanosis. A roaring continuous murmur, coarse precordial thrills, and capillary pulsation were present. Blood pressure was 160/0. Right axis deviation persisted. This finding had raised the question of associated intracardiac anomaly and resulted in hesitancy on the part of the cardiologist to recommend operation. It was now felt that operation should not be deferred any longer.

At operation the ductus was found to be equal in diameter to the aorta (10 mm.) and to enter it in a fetal direction. Pronounced cardiac irritability caused concern during operation but following slow occlusion blood pressure stabilized at 150/110. The ductus was divided and sutured. Recovery was uneventful.

Following operation cardiac size and pulmonary vascular markings progressively decreased. Six months later it was in the high normal range. Two years later it was small and normal in contour. Pulmonary vascular markings were normal. Right axis deviation was less marked but still present. Three years later all findings were normal. She was very active, growing and developing normally. No murmurs were present.

CASE 3.—I. R. At 8 months of age a heart murmur was noted, but the baby had shown poor weight gain and rapid respiration since birth. There was no cyanosis. At 3 years she was small, poorly nourished, and retarded in growth. There was a loud systolic murmur centered over the third left interspace accompanied by a thrill. X-ray showed great enlargement and prominent pulmonary vascular markings. Electrocardiogram showed tachycardia and low voltage. A tentative diagnosis of atrial septal defect was made. For 3 years she was observed at intervals. She grew slowly and remained underweight, was moderately restricted, and never showed cyanosis or evidence of failure.

On admission at 6 years she showed a rought systolic murmur in the pulmonary area with a loud P_2 and palpable thrill. Blood pressure was 110/80. Z-ray showed general cardiac enlargement and evidence of pulmonary hypercirculation. Angiocardiogram demonstrated normal sequential filling of the cardiac chambers but persistent opacification of the pulmonary artery. Cardiac catheterization showed right ventricular pressure equaling systemic, with pulmonary hypertension almost to aortic, higher oxygen content in pulmonary artery than right ventricle and slight peripheral arterial unsaturation. A diagnosis of patent ductus with balanced flow was made.

At operation a short wide ductus, 12 mm. wide and 5 mm. long, was found entering the aorta at right angles and tenting the pulmonary artery upward. It was divided using slow occlusion by a Potts-Smith-Gibson clamp around the aortic origin and a Potts ductus clamp at the pulmonary origin. Each cut end was sutured. The pressure rose from 80/50 to 120/90 on closing the ductus. There was no cardiac irregularity.

Recovery was uneventful. Six months after operation she had gained no weight but seemed well, was free of murmurs, and x-rays showed a heart of normal size with decrease in pulmonary vascular markings.

CASE 4.—E. M. A heart murmur was first noted at 5 days, and cardiac enlargement was shown by x-ray. He grew slowly, remained underweight, and suffered from repeated attacks of "asthmatic bronchitis." He was first observed at the age of 4 years. A loud systolic murmur in the fourth left interspace was heard with accentuated P_2. During the next 2 years respiratory difficulties continued and he became dyspneic on slight exertion. On 2 occasions he raised bloody sputum.

On admission at 6 years he was small and undernourished with a marked pigeon breast deformity but no cyanosis. The heart was much enlarged with a systolic heave and thrill. A loud systolic murmur was present at the base with very loud snapping P_2 and a split second sound at the apex. A diastolic murmur was present high under the clavicle but there was no continuous murmur. Blood pressure was 130/50. X-ray showed marked cardiac enlargement with a prominent pulmonary shadow and increased bronchovascular markings. Electrocardiogram was not remarkable. Cardiac catheterization was unsuccessful. Angiocardiogram showed a markedly enlarged pulmonary artery and negative shadow suggesting blood shunting from the aorta.

At operation a ductus 20 mm. in diameter at the pulmonary end and 12 mm. at the aortic end and about 10 mm. long was found. The diameter at the aorta was equal to that of the aorta which it entered at a right angle. After dissecting the ductus out, temporary occlusion caused slowing and dilatation of the heart. It was therefore decided to free the aorta in order to use a Potts-Smith-Gibson clamp. During this dissection sudden hemorrhage occurred from a tear at the pulmonary origin of the ductus. The ductus was occluded, controlling hemorrhage but again causing cardiac dilatation and disappearance of peripheral pulse. On releasing the ductus cardiac action improved with further controlled hemorrhage up to 200 c.c. The ductus was now doubly clamped with Potts ductus clamps, the aortic end was ligated under the clamp, and the wide pulmonary end was closed with a continuous suture. Cardiac action again became feeble during this procedure but improved following massage and intracardiac epinephrine. Following operation blood pressure rose to 136/90. He made a good recovery.

A year after operation an inguinal hernia was repaired without incident. Two years after operation he was hit by a car but sustained no serious injury. Three years after

operation a tonsillectomy was performed. In this period he gained 6 inches in height and 14 pounds in weight. His heart has remained free of murmurs and x-rays show normal heart size. Blood pressure is stable at 125/80.

CASE 5.—E. C. A heart murmur had been known to be present since birth. As a baby she ate poorly and gained slowly, but during her second year improved. At 4 years examination showed a small but well-nourished child with no cyanosis. A rough systolic murmur centering over the third and fourth left interspaces was transmitted faintly to the back. X-ray showed an enlarged heart with increased pulmonary vascular markings. Two years later the findings were unchanged; she continued lively and well. Over the next 4 years she gradually became more and more delayed in growth and tolerated exercise less and less well.

On admission at 10 years she was a small, slightly obese sedentary child, not cyanotic. The loud systolic murmur centering over the fourth interspace, accentuated P_2, and obvious cardiac enlargement were unchanged. Blood pressure was 120/80. X-ray continued to show cardiac enlargement and heavy pulmonary vascular shadows. Electrocardiogram showed abnormalities suggesting myocardial disease. Cardiac catheterization revealed a pulmonary arterial pressure of 75/50 at a time when aortic pressure was 110/66. Pulmonary arterial oxygen was increased over that in the right atrium and ventricle and aortic oxygen saturation was 87 per cent of capacity. The catheter passed through the ductus into the descending aorta.

At operation a dilated pulmonary artery and a short wide ductus about 8 mm. in diameter and 4 mm. long were found. Division between Potts ductus clamps was accomplished after temporary occlusion was found to result in a rise in systemic pressure from 90/70 to 140/110. After suture of the aortic end, difficulty was encountered in suturing the pulmonary end of the ductus because sutures tore through the dilated pulmonary artery wall. Closure was effected after some minor hemorrhage by using a purse-string suture including pericardial reflection, supplemented by a pledget of Gelfoam.

Postoperatively she did well for 3 days. She then began to run an increasing fever without objective signs. Antibiotic therapy was changed from penicillin-streptomycin to erythromycin without effect. Fever reached 105° F. on the sixth day, she developed diarrhea and died suddenly during defecation early on the seventh day.

At autopsy she was found to have a pulmonary embolus from an undetermined site. There was suppuration in the mediastinum at the site of the Gelfoam pledget and in the pericardial sac; the organism was a staphylococcus resistant to all antibiotics. The ductus closures were healing; there was no cardiac lesion other than hypertrophy. The pulmonary vessels showed intense hyperemia without definite intimal changes of the type associated with pulmonary hypertension.

CASE 6.—T. Y. For the first 7 or 8 months of life almost constant cyanosis was present. He ate poorly, gained and grew slowly. Many attacks of respiratory infection and tonsillitis occurred but tonsillectomy was refused because of the murmur. Cyanosis became intermittent, occurring on exertion, with considerable dyspnea.

On admission at 12 years he was poorly developed and nourished, weighing 60 pounds, showing no cyanosis. A loud harsh systolic murmur and thrill were heard at the base. No diastolic murmur was present. Blood pressure was 124/0. X-ray showed a grossly enlarged heart and prominent pulmonary artery. Electrocardiogram was not remarkable.

At operation a ductus 1 cm. long and 2 cm. in diameter was found. During dissection brisk bleeding from the posterior wall of the ductus occurred which was controlled by tying the ductus with a single ligature and placing a piece of muscle between ductus and bronchus. Blood pressure rose to 160/90 on closing the ductus, falling to 140/90 at the close of the procedure.

Postoperatively 400 c.c. of blood were aspirated from the left pleural cavity on the second day. On the fifth day a large epistaxis occurred. Blood pressure fell to 118/68. No murmurs were present until the sixth day when a continuous murmur was heard. Thereafter inconstant systolic and diastolic murmurs were heard, and by discharge 2 weeks later a definite continuous murmur was present with blood pressure 112/40. The continuous murmur

persisted and peripheral pressure slowly changed from 110/40 one month after operation to 110/20 two months after and 96/0 three months after. Four months after operation he was readmitted. Now he showed daily fever to 102° F. and repeated positive blood cultures. He was treated with sulfathiazole without effect for a period of 4 weeks.

At the second operation no trace of the previous ligature was found. The ductus was smaller and thicker walled (1 cm. in diameter) than on the first attempt. It was doubly ligated with umbilical tape without incident. Blood pressure rose to 120/80 after ligation.

Postoperatively all blood cultures remained sterile, though he remained febrile due to atelectasis for the first week. He recovered slowly and was discharged 2 months after operation. At this time there were no murmurs, peripheral pressure was stable at 120/90, and x-ray showed significant decrease in cardiac size and clearing of the lung fields.

He has remained well for the following 15 years. He served in the Army in Germany in World War II and has subsequently married and is employed as a mechanic without physical disability. Examination now shows him to be a well-developed adult of small stature. There are no murmurs. Blood pressure is 125/80. X-ray shows normal cardiac size and contour and clear lung fields. Electrocardiogram shows no abnormality.

CASE 7.—L. U. She was first seen by a cardiologist at the age of 5 years. A continuous murmur, great cardiac enlargement, and large pulmonary artery shadows by x-ray were noted. Throughout childhood her activity was limited by dyspnea. At 20 she married and became pregnant. Following delivery her symptoms became worse and her second pregnancy was difficult. A third pregnancy at 25 was interrupted therapeutically. Decompensation occurred 3 years later but responded to digitalis, bed rest, and mercurials. At 30 decompensation again occurred and fibrillation began.

On admission at 30 years of age she was thin but normally developed with no cyanosis. Her heart was enlarged with a diffuse heaving apical impulse in the mid-axillary line, fibrillating at 90 to 100. Blood pressure was 150/40. In the pulmonary area a loud harsh systolic murmur was present; at the apex rough systolic and low-pitched mid-diastolic murmurs were heard. X-ray showed a grossly enlarged heart with markedly enlarged pulmonary artery. Electrocardiogram showed left ventricular hypertrophy, auricular fibrillation, and myocardial disease. At cardiac catheterization the catheter passed through the ductus into the descending aorta. Pulmonary pressure was 75/30 and aortic pressure was 112/45. Arterial saturation was 96 per cent, and pulmonary flow was five times peripheral flow.

At operation a short wide ductus, 8 mm. long and 15 mm. in diameter, was found. The aorta and ductus were mobilized and temporary closure was followed by a rise in peripheral pressure to 160/90. The ductus was now slowly clamped, divided, and sutured. Difficulty with suture line bleeding required additional sutures and a Gelfoam strip was placed between the suture lines.

Postoperatively peripheral pressure remained elevated to 150/84. She recovered well, all murmurs disappeared. Attempts to convert her to regular rhythm with quinidine were unavailing. One month later cardiac and pulmonary size had regressed markedly. Three years later she remained well without digitalis.

CASE 8.—V. M. A heart murmur was noted at birth, but infancy and childhood were apparently not much handicapped. She grew and developed normally, and attended regular school through high school with only slight exertional dyspnea. At 15 years the diagnosis of patent ductus arteriosus was made, and operation was performed in another hospital. The ductus was exposed and found to be large, thin-walled, and tense. During dissection hemorrhage occurred and the procedure was terminated without closure of the ductus. She recovered, completed school, took a job as a machine tool operator, and married. At 25 decompensation occurred, but she recovered rapidly on digitalis and restriction of activity. Three months later she became pregnant and therapeutic abortion was carried out. At this time cardiac catheterization showed a pulmonary arterial pressure of 88/55 with aortic pressure of 105/60 and arterial saturation of 87 per cent.

On admission at 25 she was a small, poorly nourished but normally developed woman who looked pale rather than cyanotic. Left vocal cord paralysis was present. There was a

loud, rough systolic murmur at the base with a softer diastolic murmur and accentuated P_2. X-ray showed gross cardiac enlargement with prominent pulmonary vascular markings. Electrocardiogram showed both right and left ventricular hypertrophy and myocardial disease. Blood pressure was 105/60.

At operation a ductus 20 mm. in diameter and 5 mm. long was found. The aorta was enlarged and atheromatous. The aorta was mobilized and the ductus dissected out with difficulty because of surrounding fibrosis. Ultimately the ductus was divided between Potts ductus clamps, and the pulmonary stump was sutured without incident. Closure of the aorta by suture was unsatisfactory and resulted in hemorrhage due to the presence of an atheromatous plaque at the junction of ductus and aorta. A purse-string suture through the aortic adventitia was therefore used, and no further bleeding occurred. During this maneuver peripheral pressure fell to 80/60 and the heart rate became slow and irregular. A transfusion of 1,500 c.c. was given.

Postoperatively she remained hypotensive with a heart rate of 140. Transfusion was continued without effect. Noradrenaline was given intravenously but peripheral pulse became imperceptible, then respirations ceased. The wound was reopened and the heart found dilated and beating feebly. There was no evidence of further bleeding. In spite of massage and intracardiac epinephrine the heart failed to respond.

At autopsy the ductus closure was intact. About 300 c.c. of blood was present in the pleura. There were many old pleural adhesions. Atheromatous plaques were present in the aortic arch and main pulmonary artery. The smaller pulmonary arteries showed diffuse intimal thickening. The heart was generally hypertrophied and dilated but showed no valvular lesions or anomalies of development.

CASE 9.—N. Z. Early in childhood the presence of a heart murmur was recognized and physical activity was restricted. She was always small and thin but developed normally and was able to go to school and carry out normal activity short of vigorous exercise. She was never cyanotic. She became a secretary and had no untoward symptoms until the age of 29 when she had the first of a series of episodes of palpitation. She had no dyspnea, cyanosis, or ankle edema. At 31 she was put on digitalis and the attacks were controlled with quinidine.

On admission at 34 years she was small, thin but well developed, and showed no cyanosis dyspnea or orthopnea. There was a left kyphoscoliotic deformity of the thoracic spine. The heart was enlarged with apical impulse in the sixth interspace, anterior axillary line. A coarse thrill and loud, rough systolic murmur was present throughout the precordium, the murmur was heard best over the apex where a short high-pitched diastolic murmur was present. P_2 was accentuated. Blood pressure was 150/20. X-ray showed marked enlargement of both ventricles and rotation of the heart. Pulmonary vascular shadows were prominent. Electrocardiogram showed left ventricular hypertrophy and evidence of myocardial disease. A pronounced Corrigan's pulse was present. Cardiac catheterization showed a pulmonary artery pressure of 113/60 when systemic pressure was 137/59. There was increased oxygen content of pulmonary arterial blood as compared to the right atrium and ventricle. Arterial oxygen saturation was 91 per cent and decreased equally in brachial and femoral arteries to 56 per cent on breathing 10 per cent oxygen. Retrograde aortogram showed simultaneous filling of the aortic arch and a much dilated pulmonary artery.

At operation a ductus 20 mm. in diameter and 15 mm. long was found. The pulmonary artery was greatly dilated and tense. The ductus was carefully dissected by mobilization of the aorta. Temporary slow occlusion caused a fall in systemic pressure from 140/40 to 120/80, but there was no cardiac dilatation or irregularity. The ductus was slowly closed with a Potts-Smith-Gibson clamp and divided between this and a Glover clamp on the pulmonary origin. Each end was closed by arterial suture without hemorrhage and a pledget of Gelfoam was placed between the sutured ends. A lung biopsy was taken from the lingula of the left lung.

In the first 24 hours after operation systemic pressure rose from 140/80 to 180/110. Two days later it was 160/90 and by discharge 13 days after operation it had stabilized at 140/90. There were no complications of healing, and no murmurs could be heard.

Thereafter, she continued well. One year after operation she had gained 8 pounds and was back at work with no further symptoms of palpitation or fatigue. No digitalis was taken after discharge. X-ray showed significant decrease in cardiac size and disappearance of increased pulmonary vascular markings. Blood pressure was 130/90, and there were no murmurs. Section of the lung showed no significant arteriolar thickening.

CASE 10.—G. C. A heart murmur was noted from birth but she was not cyanotic and grew and developed normally. Dyspnea at play was first noted at 3 years. Cyanosis with exertion or fatigue or during cold weather was noted from the age of 7 years. Thereafter she was severely restricted in activity. At 17 years she had an episode of chest pain and dyspnea following an appendectomy after which she was kept in bed for 7 months and on much restricted activity for the following 2 years. There was no fever and no signs of failure were present at this time. Thereafter she did well, working as a secretary. At 24 years a severe hemorrhage followed tonsillectomy. At 28 she married. At 29 years she developed fever, chest pain, cough, and peripheral edema, which improved after 6 weeks of bed rest but recurred 6 months later. She was hospitalized, treated with digitalis mercurials and large amounts of penicillin, and recovered after 2 months.

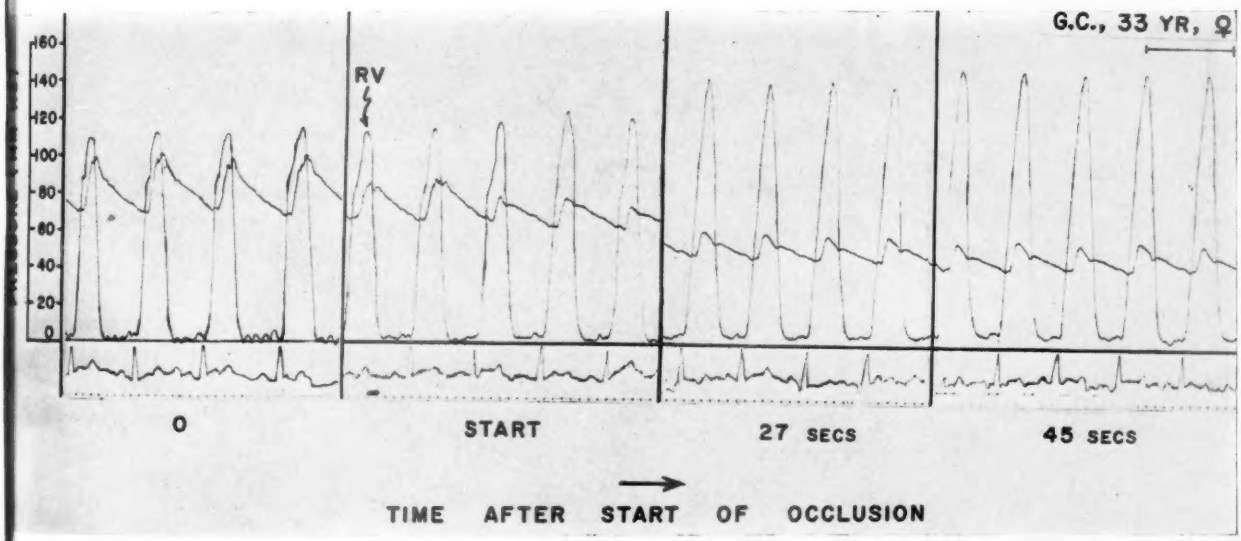

Fig. 1.—Tracings at operation in Case 10 before and at intervals following occlusion of the ductus. Right ventricular and left subclavian (aortic) pressures are superimposed on the same scale. Aortic systolic pressure nearly equals right ventricular before occlusion but falls progressively over 45 seconds. Right ventricular systolic rises, and end diastolic rises very slightly. The pressures at 45 seconds remained stable for 5 minutes thereafter, giving hope that ductus closure would be tolerated.

At 30 years she was admitted for study. She appeared plethoric but not cyanotic with hematocrit of 63 per cent and hemoglobin 17 Gm. A harsh systolic murmur was present in the pulmonary area with a softer diastolic murmur along the left sternal margin. P_2 was much accentuated. The heart was enormously enlarged primarily to the right as shown by x-ray. Electrocardiogram confirmed marked right ventricular hypertrophy. Cardiac catheterization was attempted but discontinued because of the appearance of right bundle branch block as the catheter entered the right ventricle. For the next 3 years she continued with increasing dyspnea on exertion and more evident cyanosis. She was bedridden much of the time. At 31 she was readmitted and cardiac catheterization was done. Pulmonary arterial and aortic pressure were identical at 123/83 and the catheter passed from pulmonary artery into the descending aorta. Oxygen saturation was 88 per cent. At 32 she became pregnant and therapeutic abortion was carried out. Her physical signs were unchanged. Hematocrit was 69 per cent and she was in borderline failure. She recovered and remained in fair health for the following year.

TABLE I. SUMMARY OF CLINICAL FINDINGS

CASE	AGE	MURMUR	EKG	SPECIAL STUDIES	ANATOMY	OPERATIVE COURSE	RESULT	PATHOLOGY
1. M. A.	1½	Atypical	Heart block	Reversal by angiogram	15 mm. in fetal position	Slow closure, uneventful, pressure fell	Died 2 months	I.V.S.D. pulmonary vascular changes
2. S. G.	2	Typical	Right axis	None	10 mm. in fetal position	Cardiac irregularity, slow closure, pressure fell	Well 3 years	—
3. I. R.	6	Atypical	Right axis	P.A. 66/46 A. 70/40 90% sat.	12 mm. in usual position	Slow closure, uneventful, pressure rose	Well 6 months	—
4. E. M.	6	Atypical	Normal	Angio. showed L-R shunt	20-12 mm. in usual position	Hemorrhage, cardiac massage, pressure fell	Well 3 years	—
5. E. C.	10	Atypical	Abnormal, no axis shift	P.A. 75/50 A. 110/66 87% sat.	8 mm. in usual position	Minor bleeding, pressure rose	Died 7 days	Pulmonary embolus; no significant vascular changes
6. T. Y.	12	Atypical changing to typical after partial closure	Normal	None	20 mm. in usual position	1st Op. hemorrhage, single ligature, recurrence + S.B.E. 2nd Op. double ligation, uneventful	Well 16 years	—
7. L. U.	30	Typical changing to atypical	Left axis	P.A. 75/30 A. 112/45 96% sat.	15 mm. in usual position	Slow closure, uneventful, pressure rose	Well 2 years	—
8. V. M.	25	Atypical	Rt. + L.A. hypertrophy	P.A. 85/55 A. 105/60 87% sat.	20 mm. in usual position	1st Op. hemorrhage, no closure; 2nd Op. hemorrhage, pressure fell	Died 3 hours	Pulmonary vascular changes
9. N. Z.	34	Atypical	Left axis	P.A. 113/60 A. 123/83 91% sat.	20 mm. in usual position	Slow closure, uneventful, pressure fell	Well 1 year	No significant vascular changes
10. G. C.	33	Atypical	Right hypertrophy	P.A. 123/83 A. 123/83 88% sat.	30 mm. in usual position	Pressure fell with occlusion, fatal hemorrhage	Died at operation	Marked pulmonary vascular changes

On admission at 33 she was plethoric and cyanotic with greater cyanosis and clubbing of the toes. Hemoglobin was 20 Gm. and hematocrit was 71 per cent. Physical signs, x-ray, and electrocardiogram showed no changes. Phlebotomy was attempted but was unsuccessful due to prompt clotting. It was decided to proceed with an attempt to close the ductus, performing phlebotomy at operation and controlling the procedure by concomitant pressure studies with the intention of refraining from final closure if evidence of right ventricular failure occurred with temporary occlusion.

At operation continuous pressures were recorded from right ventricle and left subclavian artery. The ductus was found to be 30 mm. in diameter and 25 mm. long. The pulmonary artery was greatly dilated and tense; the aorta was not dilated and the pulmonary artery overrode it. The aorta and ductus were dissected free and an attempt was made to place a Potts-Smith-Gibson clamp. Because of the great width of the ductus and its proximity to the left subclavian origin, this proved impossible. The ductus was slowly occluded with a Glover clamp while pressures were observed. (Fig. 1.) The ductus was released and dissection was carried proximally over the pulmonary origin in order to obtain a sufficient cuff for suture. In so doing a tear occurred at the junction of ductus and pulmonary artery. Clamps were immediately applied, controlling the aortic end satisfactorily. The pulmonary tear, however, extended beyond the clamp blade and profuse hemorrhage ensued. Further attempts to control bleeding were defeated by the friable pulmonary arterial wall which repeatedly tore out of controlling clamps. The patient died of exsanguinating hemorrhage.

At autopsy marked hypertrophy of the right ventricle was present. The pulmonary artery was markedly dilated and atheromatous at the site of laceration. All major pulmonary arteries showed thickened atheromatous walls. Many of the smaller arteries and arterioles were blocked by intimal thickening or organized thrombi, and the changes in those which remained patent were those characteristic of advanced pulmonary hypertension. There were no other intracardiac defects.

DISCUSSION

Because of the progressive deterioration of patients with atypical ductus and the disastrous effects of reversal of flow which may occur late in the syndrome, it is manifestly important to detect and correct the lesion early in life. The diagnosis, however, is difficult. Only one of the patients (Case 2) had a characteristic machinery murmur, and in her case the presence of right axis deviation on electrocardiogram led to delay because of suspicion of another lesion. Three others (Cases 3, 8, and 10) also showed right axis deviation; in 2 (Cases 7 and 9) there was left axis deviation in spite of definite pulmonary hypertension; 2 others showed other abnormalities; and in 2 the electrocardiogram was not remarkable. The clinical diagnosis of septal defect was made in 4 of the 6 children (Cases 1, 3, 4, and 5), and the diagnosis of patent ductus in these was revealed only by angiocardiography or cardiac catheterization. Only in Case 1 was a septal defect also present. Two of the 4 adults (Cases 7 and 9) were considered to have rheumatic heart disease for many years, and a third (Case 10) was thought to have an atrial septal defect. Yet all of the patients were known to have had murmurs since infancy. The importance of carrying out definitive diagnostic studies in all such patients is, therefore, obvious.

How important it is to recognize this syndrome before it has reached the reverse flow phase is well emphasized by the discouraging reports of surgical closure attempted after it has appeared. A review of the recorded experience

to date, including ours, shows that 45 have undergone surgery.[1, 2, 3, 9, 13-21] Of these,[23, 24, 25, 27] 23 survived operation (one died 7 months later), 17 died during or after operation, in 4 the operation could not be completed because of cardiac arrest or hemorrhage, and in 1, the ductus was only partially occluded. However, 4 of the survivors had associated intracardiac defects which might tend to ease the burden of duct closure, while one who died was found at autopsy to have fibroelastosis and mitral and aortic stenosis. Approximately one-third of the failures were due to technical errors.

Physiologic Considerations.—The first two patients were infants whose ductus retained its fetal equality in size to the aorta and its fetal obliquity. Although we have no data to prove reversed flow in Case 2, this was definitely demonstrated in Case 1 where a ventricular septal defect added a left-to-right shunt at the ventricular level (Fig. 2). The presence of right axis deviation, the cardiac irregularities, and the fall in systemic pressure on closure of the ductus suggest that it may also have been present in Case 2. This type of reversal may well be related to the obliquity of entrance, although it is of interest that vascular changes were present in the pulmonary vessels in the first case. In both, gradual closure of the ductus was successful and it seems unlikely that altered hemodynamics related to ductus closure were related to death in Case 1.

None of the next 4 children showed reversal when studied under anesthesia, but the finding of arterial unsaturation on 2 (Cases 3 and 5) and the occurrence of cyanosis on exertion in Case 6 indicate a pressure relationship of "balance" in which reversal occurred during some phases of the cardiac cycle or under conditions of stress. Case 8 showed similar findings. In these patients there is a marked increase in circulating volume, and the heart may not tolerate the strain thrust upon it by sudden closure. As demonstrated by Case 4, loss of blood may be a lifesaving factor. This was also probably a factor in Case 6 though it led to inadequate closure on the first attempt. The ductus lumen was smaller at the second procedure, and this was well tolerated. Case 8 demonstrates the disastrous result of injudicious blood replacement under these circumstances. In Cases 3 and 5 slow closure was successful, and the immediate rise in systemic pressure indicated that the heart in these cases was equal to the load put upon it.

Patients 7 and 9 were adults whose pulmonary pressures were high but whose left ventricles remained dominant, maintaining normal saturation at rest. In both, careful slow occlusion was well tolerated, though the immediate fall in systemic systolic pressure in Case 9 followed by a period of several days of hypertension indicates that judicious phlebotomy might have added safety to the procedure. It is interesting that pulmonary arteriolar thickening was not present in this patient in spite of great elevation of pulmonary pressure, whereas it had occurred in Case 8 when pressure was lower.

Case 10 is the only case in which definite reversal of flow was an established fact. This patient showed marked obstruction to pulmonary flow by pulmonary vascular thickening and thrombosis. Although her arterial unsaturation at rest was no greater than in Case 5 or Case 8, the presence of marked

polycythemia, cyanosis, and toe clubbing indicated a larger volume of reversed shunt under ordinary conditions. Unquestionably a reduction in polycythemia would have made the procedure safer, but it may be questioned whether successful closure could long have been tolerated by the right ventricle.

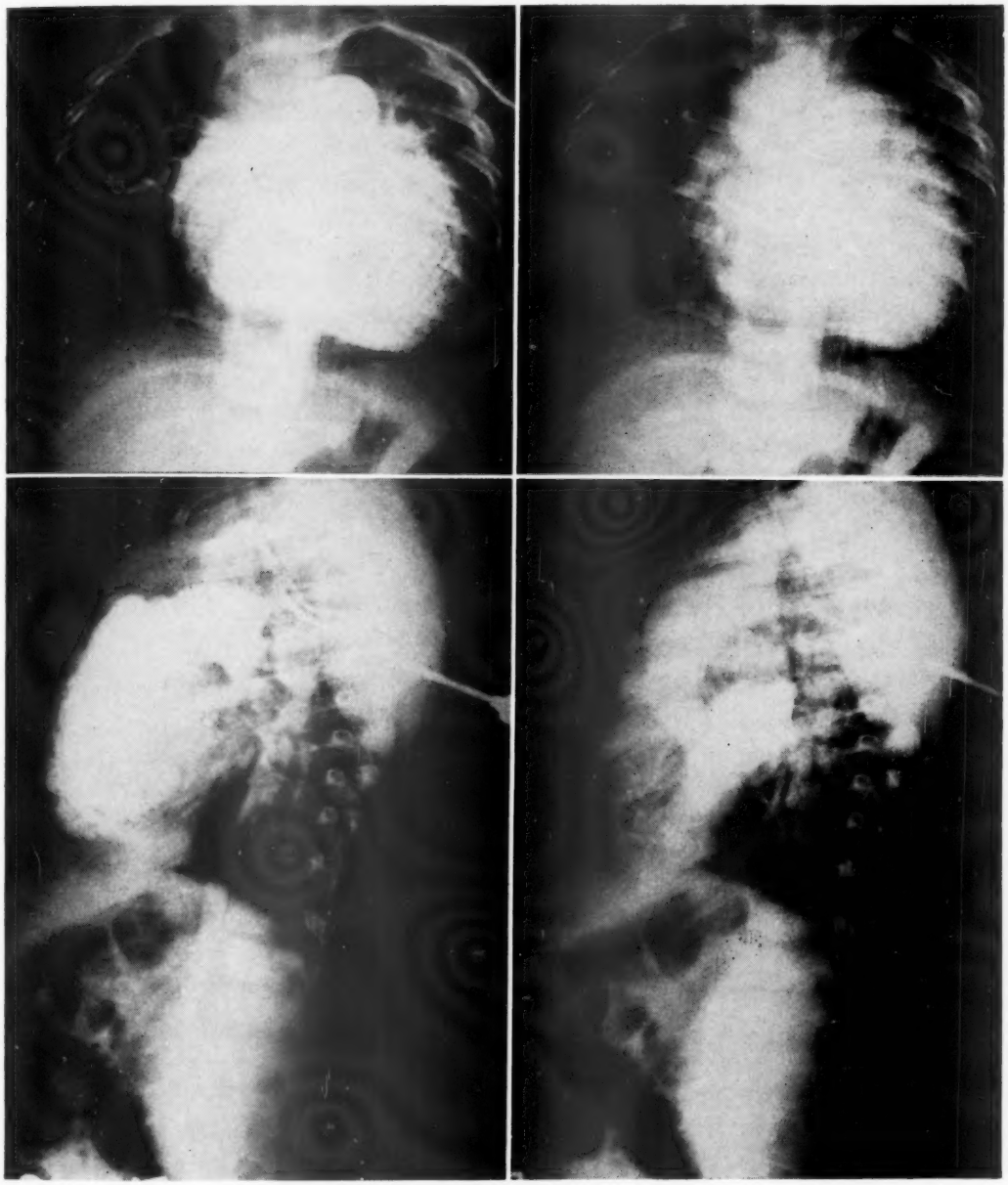

Fig. 2.—Angiocardiograms on Case 1. At 1.5 seconds (left) simultaneous filling of right ventricle, pulmonary artery and descending aorta is seen. At 5 seconds (right) the left ventricle, ascending aorta and refilling of the pulmonary artery is demonstrated.

It is evident that within this group there is a wide variation in the actual degree of pulmonary vascular resistance. It is probable that in some patients closure of the ductus brings to light a resistance which greatly exceeds that of

the systemic circulation. In such a situation a burden falls on the right ventricle which it cannot carry. On the other hand, if the anatomic resistance is less, slow closure of the ductus, giving time for peripheral circulatory adjustment, aided by judicious reduction of total circulating volume, may permit successful closure. As pointed out by Kjellberg and associates,[19] the presence of a patent ductus in such patients would increase rather than decrease the work of the right ventricle, for whatever volume of blood is shunted from the pulmonary artery through the ductus, the systemic circulation is increased an equal amount. This in turn increases the output of the right ventricle. In other words, the right ventricle (like the role of the left ventricle in the typical case of patent ductus arteriosus) must support the blood flow through the lungs as well as that recirculating through the systemic circulation via the shunt. Therefore, it would seem reasonable to advise operation for those cases in which the resistances of the two circulations are approximately equal, that is, where there is 'balanced flow' or where there is reversal but a large left-to-right component still remains ('mixed flow').[9, 14, 19, 22] A number of patients have been reported whose pulmonary hypertension has receded to normal levels several months following ductal closure.[1, 6] It seems probable that this has also occurred in the successful cases in this series. But the extent of anatomic vascular change in these is uncertain and its regression cannot be demonstrated. A similar histologic picture, found in certain patients with pulmonary hypertension secondary to mitral stenosis, however, has been noted to regress after surgical relief.[10]

Technical Considerations.—The death of 4 of the 10 patients emphasizes the high risk of operation in this group. In 2 (Cases 1 and 5) apparently successful closure was accomplished. Case 1 suffered from heart block related to the associated ventricular septal defect, and death followed convulsions associated with disturbances of cardiac rhythm. Although these had been observed before operation, their more violent occurrence in the immediate postoperative period with death in convulsions 2 months later suggests that closure of the ductus played some part in increasing the fundamental abnormality. Case 5 died of a pulmonary embolus associated with sepsis, and the latter was related to difficulties with hemostasis at operation.

Major hemorrhage occurred in 4 patients (Cases 4, 6, 8, and 10) and resulted in death in the last. It is evident that the high pulmonary pressure and vascular wall changes make this a special risk. For this reason a posterolateral incision with complete mobilization of the aorta and gradual occlusion of the aortic origin with a Potts-Smith-Gibson clamp, as recommended by Conklin and Watkins,[5] is a real safeguard. In this manner the aortic end of the ductus can be cut flush with the aorta and closed with a linear suture, leaving a good cuff of ductus wall for suture of the more dangerous pulmonary end.

Gradual occlusion of the ductus also can best be controlled by this technique. If direct pressure readings from right ventricle and aorta are obtained during operation, the surgeon can best judge the degree of pulmonary

resistance and the capacity of the right ventricle to overcome it. A rising ventricular end diastolic pressure foretells imminent failure, and a sustained high ventricular systolic pressure with an accompanying low aortic systolic indicates an imbalance which cannot long be tolerated. Even without direct pressure recording, this situation can be judged to be present if the systemic pressure falls instead of rises when the ductus is closed (Cases 1, 2, 4, 8, and 9).

It appears paradoxical to resort to phlebotomy to control systemic hypotension, yet Case 4 illustrates that controlled blood loss may be lifesaving under these circumstances, and Case 8 illustrates the fatal result of transfusion. Preoperative phlebotomy may be wise when the situation is anticipated from the finding of "balanced" flow on catheterization. It is especially important in reducing the polycythemia of reversed flow which adds an extra factor of resistance to flow through narrowed pulmonary channels.

With these safeguards the procedure should succeed more often than it has in the past. Yet even with them the risk will be much greater than is the case when the ductus is small. The risk must be accepted, however, for progressive deterioration is otherwise inevitable.

SUMMARY

A series of 10 patients with atypical patent ductus arteriosus and varying degrees of pulmonary hypertension is reported. These show, as do similar cases reported by others, that there is great variation in the physiologic implications of the syndrome. All show physical signs which differ from those typically associated with the anomaly and which tend to obscure or confuse the diagnosis. These are related to the size of the ductus and resulting pulmonary hypertension and varying degrees of balanced or reversed flow.

Although it is shown that in general pulmonary hypertension increases with age, clear-cut progressive pulmonary vascular changes are not demonstrated. The majority showed predominantly left-to-right flow, but varying degrees of balance or reversal were demonstrated in 6 and presumably present in others; only one showed continuous reversal with cyanosis.

Physiologic response to closure is discussed. It is shown that gradual closure, judicious phlebotomy, and technical details of closure to guard against the increased risk of hemorrhage can lower the present risks of operation in this group. With these safeguards it is concluded that closure should be attempted in all cases unless it can be demonstrated at operation that cardiac failure will result.

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Discussion

DR. DONALD B. EFFLER (Cleveland, Ohio).—I wish only to discuss a feature of the surgical treatment of atypical ductus arteriosus. At the Cleveland Clinic Foundation

we have been very much impressed by Dr. John C. Jones's use of the posterolateral approach and his suggestion to begin the dissection by mobilization of the aorta above and below the ductus before any effort is made to dissect the ductus itself. We have found this to be very helpful in reducing the threat of operative hemorrhage.

We have operated upon a series of 32 infants with atypical ducti with absence of the continuous murmur, or some other abnormality not consistent with the usual ductus pattern. In every case it has been possible to divide the ductus and perform the operation without an operating room fatality. One patient died in the hospital of a pulmonary complication.

Among the adult patients there are other types of atypical ductus. We consider recannulized ducti that have previously been subjected to ligation and continuity as surgical problems of potentially increased magnitude. Other forms of atypical ductus include those with frank vascular degeneration, aneurysmal formation, and shunt reversal of varying degree. In all the adult forms of atypical ductus, we employ the posterolateral approach and the addition of controlled arterial hypotension. Hypotension is created on the table by either arterial bleeding, or the use of drugs such as Arfonad. Hypotension properly employed is a safe adjunct to surgery and is extremely valuable since it reduces the hazard of uncontrolled hemorrhage on the table, facilitates difficult dissection by literally decompressing the aorta and aneurysmal ductus, and may appreciably shorten a long, tedious dissection. Experience over the past 7 years leads us to believe that the posterolateral approach is best for all types of ductus surgery, and that controlled hypotension is an extremely valuable adjunct particularly in the complicated or atypical adult forms of the disease.

DR. JOHN C. JONES (Los Angeles, Calif.).—There are just two points I would like to make.

Hypothermia has been a great help to us in the high pressure ducti and atypical ducti, but long before we had hypothermia we observed a technique on these patients who have a high pressure ductus that I think is important to employ. The older children and the adults who have high pressure ducti usually have a good deal of sclerosis at the site of the ductus in the wall of the aorta, so we routinely treat these with a certain technique.

We clamp the aorta above the ductus, clamp the ductus near the pulmonary artery, and then clamp the aorta below the ductus. Then we carve off the ductus from the side of the wall of the aorta, taking with it the sclerotic plaque. If you try to divide this type of ductus, the clamp will frequently go through the wall and you have a miserable situation and considerable blood loss. With our three-clamp technique we have a hole in the side of the aorta and a long cuff on the ductus side. We bring the two clamps on the aorta close together and immediately suture the aorta defect transversely. The aorta clamps are removed in less than 15 minutes to establish circulation before the ductus cuff is closed with a running suture. Having used this technique for several years, we have had no hemorrhage or complication in the entire group so treated.

DR. FRANK GERBODE (San Francisco, Calif.).—I want to compliment Dr. Humphreys on his series and would like to comment on two aspects of the problem. One is the diagnosis of atypical ductus in infancy. A lot of babies get into trouble very early in life from a simple straightforward ductus. It is a curious thing this has not been really apparent to the general medical profession until quite recently. It is sometimes very difficult to make a diagnosis without special studies, and we have found in our clinic that a retrograde brachial aortagram is a safe method of studying these children and often will lead one to a diagnosis. We have employed this technique in over a hundred cases of suspected ductus in babies and found 25 per cent of them have a defect that can be corrected by operation.

I want also to mention the advisability of measuring intra-arterial blood pressures in the operating room. If there is any doubt as to whether or not the ductus is reversed,

the pulmonary artery pressure should be measured with the ductus open and closed. If the pressure in the pulmonary arteries rises when the ductus is closed, the operation should be abandoned as closure of the ductus would result in a fatal issue.

DR. GEORGE H. Humphreys (closing).—I wish to thank the discussers. We are in hearty agreement with the posterolateral approach. This is, we think, one of the factors of safety which, as pointed out by Dr. Jones, is an important one in dealing with any ductus, especially those in this category.

I had meant to make the point that Dr. Jones emphasized—that with the high pressure pulmonary circuit, the incision should be made flush with the aorta, leaving a good cuff on the pulmonary side for closure. We have not used this longitudinal cutoff and transverse repair. This sounds like a very helpful procedure, but we have usually done it longitudinally when we used that technique.

Dr. Scott raised the question of reversibility of pulmonary arterial changes. We have no proof as to whether they are reversible or irreversible. Certainly, the changes shown in this last patient look so profound that it would be hard to imagine their reversing completely. Still they might well reverse sufficiently to reduce at least to a reasonable extent the obstruction to the pulmonary flow. We have reason to believe that some reversibility is present. Certainly, there have been a sufficient number of patients catheterized after pulmonary hypertension has been demonstrated and relieved by surgery to know that pulmonary hypertension can recede. This does not mean necessarily that histologic changes have receded, however, but I believe that they probably are, in their early phases at least, reversible.

The infant of a year and a half, at autopsy showed definite evidence of these pulmonary changes; yet this patient did not die of pulmonary hypertension. He had survived his operation and would have continued to survive it had he not had a cerebral episode which resulted fatally.

I think this answers Dr. Gerbode. Certainly, in infants, I would expect a reversibility in the pattern. The use of hypothermia, I am sure, is helpful. We have not used it, nor have we used hypotension. I would be afraid to use it, but if it has proved useful in other hands perhaps I have an unfounded fear.

REPAIR OF INTERATRIAL SEPTAL DEFECTS BY A MODIFIED SONDERGAARD TECHNIQUE (CIRCUMCLUSION)

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IN THIS dawn of open heart surgery we might be considered somewhat archaic to advocate a closed method of managing interatrial septal defects. However, the safety, simplicity, and effectiveness of the technique of modified circumclusion has prompted us to relate our experiences, while re-emphasizing the basic principles enunciated by Sondergaard,[23] by Björk and Crafoord,[4] and by Cooley.[7]

In 1952 Sondergaard demonstrated the value of employing the cleavage plane between right and left atria for closure of experimentally produced interatrial septal defects. The suture, as initially employed, was anchored at the base of the aorta at one end and behind the inferior vena cava at the other, was extracardiac, and was not completely circumferential. Defects encountered clinically were not always completely closed by this technique. Subsequently, Björk and Crafoord[4] described a technique for passing a large curved needle into the antero-inferior septal rim from a position adjacent to the root of the aorta behind the right coronary artery and even into the upper border of the interventricular septum, when this was necessary. The upper end of the suture was then passed around behind the superior vena cava and tied down in the interatrial cleavage plane as a circumferential purse string. Although Crafoord stated that this technique could be utilized to close atrial defects even of the ostium primum type, it seems unlikely that this can be done safely except when the endocardial cushions are well developed, a most uncommon finding.

At the cardiovascular symposium in Detroit in 1955, Sondergaard[24] described his modification of these procedures and advocated the use of a probe to establish a track in the interatrial septum through which a suture could be withdrawn for subsequent circumclusion. He pointed out that the interatrial septum is composed of two layers of muscle enclosing loose connective tissue through which a probe could easily slide.

According to Sondergaard this techinque offers a number of advantages: the closure is accomplished in a correct anatomical plane without significant distortion of either atrial chamber; the procedure is safe, simple, and rapid; no foreign bodies or sutures are introduced into the atrial chambers; the probe avoids the hazards of the curved needle technique in relation to the

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aorta and to the auriculoventricular node and bundle of His. And in patients with ostium secundum defects, it was never necessary to place sutures in the interventricular septum.

We believe that there are several other obvious advantages. The use of the probe allows the placement of the circumcluding suture immediately at the margin of the defect. When constriction is made, tension is evenly distributed along the rim of the defect, thus minimizing or eliminating any tendency for the suture to cut through. Occasionally the rim of a defect is composed of a thin veil, in which case the track will necessarily be formed in a wider circumference in more sturdy tissue. Circumclusion effects economy of tissue

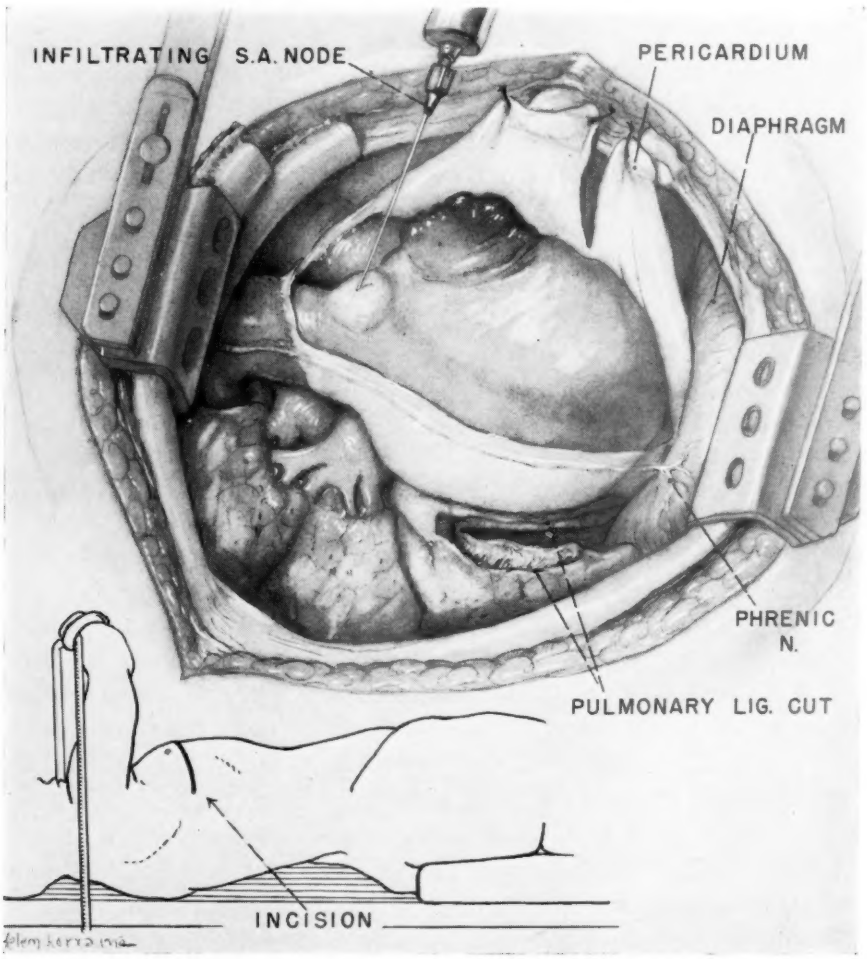

Fig. 1.—The right pleural space is entered through the fourth interspace from an anterolateral approach with division of third and fourth costal cartilages.

in the closure of large defects since the circumcluding suture lies immediately around the margin of the defect. This is true except at one point—at the interatrial muscle bundle which passes behind the ascending aorta between the right and left atrium.[21] Although it is theoretically possible to pass the circumclusion suture inside or central to the interatrial muscle bundle as a

second maneuver so that it lies truly along the margin of the septal defect at all points, we have not attempted this. And, finally, it should be pointed out that if a large needle is woven in and out of the inferior septal rim, constriction of the suture may produce some bunching of tissue and leave small residual lacunae which could go unrecognized. It is astonishing how smooth the closure feels after constriction with the circumcluding suture passed by the probe technique.

TECHNIQUE

Very satisfactory exposure is obtained by a right anterolateral approach with the patient tilted somewhat forward (Fig. 1). The pleural space is entered through the fourth interspace after transecting the third and fourth costal cartilages.

The inferior pulmonary ligament is divided and the pericardium entered anterior to the phrenic nerve with a perpendicular extension forward. The phrenic nerve is mobilized from the inferior pulmonary vein down to the diaphragm. The pericardium is split posteriorly and the oblique sinus of the pericardium is entered (Fig. 2). We have often infiltrated the region of the sinoauricular node with 1 per cent procaine, but the effect of this injection in the normothermic patient is not impressive.

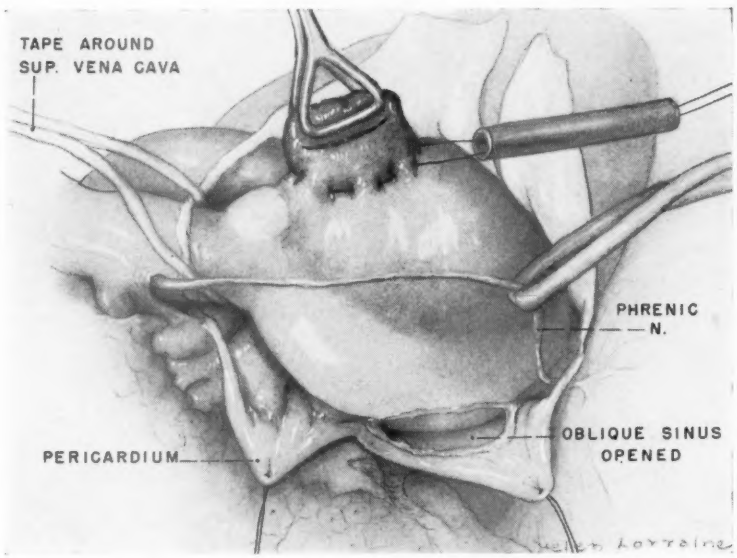

Fig. 2.—For adequate mobilization of the wall of the right atrium, the pericardial sac is split posteriorly and the oblique sinus of the pericardium is entered. We now mobilize the phrenic nerve only from the inferior pulmonary vein down to the diaphragm.

Exploration of the right atrium is next carried out through the auricular appendage. The surgeon ascertains the type and size of the defect and notes the position of all pulmonary vein orifices. The tricuspid and mitral valves are checked for stenosis or insufficiency. The rim is carefully traced around the edge of the defect, as its width superiorly and posteriorly will determine the depth to which the interatrial cleavage plane may be dissected. The relationship of the inferior margin to the inferior vena cava, the coronary

sinus, and the adjacent auriculoventricular node is noted. All ostium secundum defects, however large, are considered amenable to repair by the circumclusion technique with possible exceptions noted in the discussion.

After completing the exploration and temporarily clamping the appendage, the superior vena cava is dissected free of the pulmonary artery up to the azygos vein (Fig. 3). As the superior vena cava is lifted forward, the dissection of the interatrial groove is begun. The depth of the dissection allowed

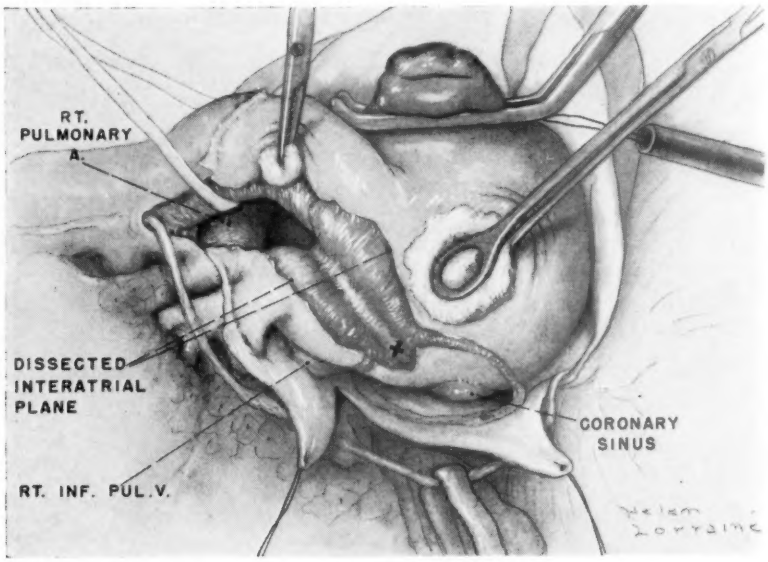

Fig. 3.—The interatrial cleavage plane is dissected anterior to the pulmonary veins. The "X" (point of emergence of probe) is marked somewhat more superior than is applicable to most cases.

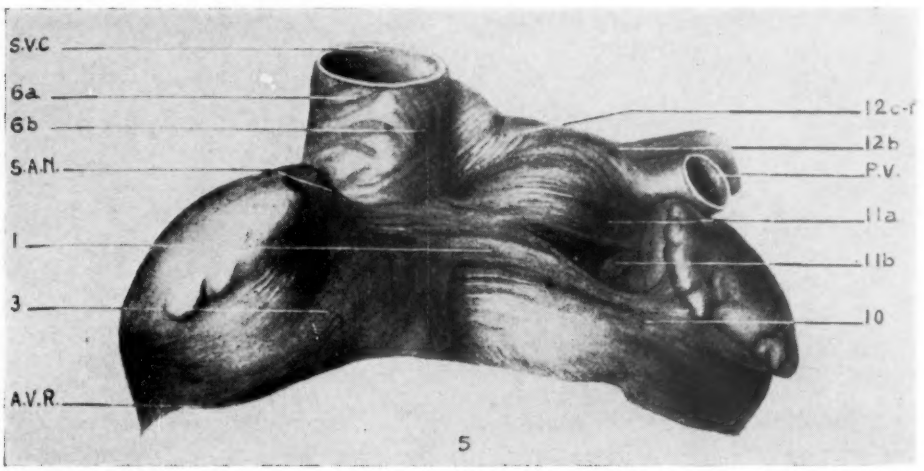

Fig. 4.—Interatrial muscle bundle (1) (Fig. 5 From Papez, Am. J. Anat. 27:283, 1920.)

varies with the size and position of the interatrial septal defect and the width of the superior and posterior rim. In general the cleavage plane is deepest behind the superior vena cava and becomes increasingly shallow toward the inferior vena cava. The dissection may be checked from time to time with

intracardiac palpation. The groove terminates anteriorly and superiorly at the interatrial muscle bundle, and we have not attempted to dissect central to this structure (Fig. 4). We have found it unnecessary to encompass the inferior vena cava with a tape except in those cases in which the lower margin of the defect extends down to the region of the inferior vena cava. In these instances, elevation of the inferior vena cava facilitates the emergence of the probe.

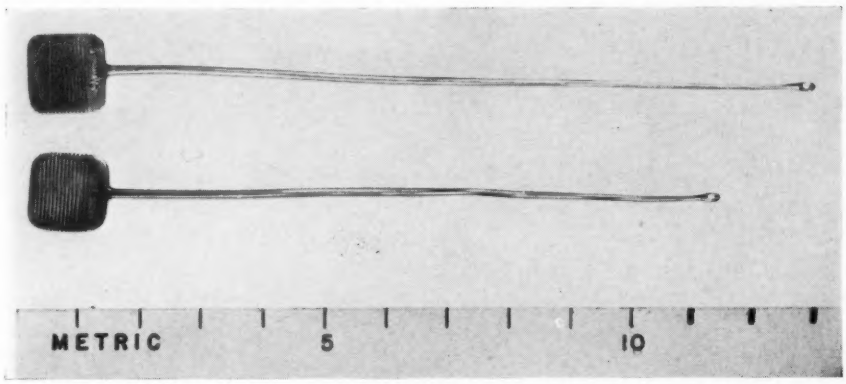

Fig. 5.—Short flexible probes with smooth tip and eye which facilitate passage of instrument and withdrawal of circumcluding suture.

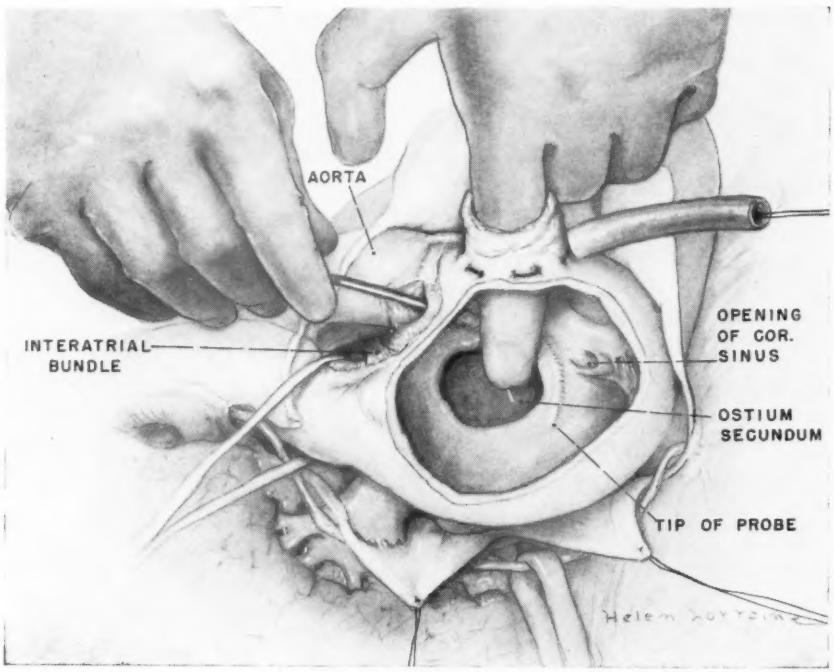

Fig. 6.—The probe is inserted into the interatrial septum at the root of the aorta and guided with the finger.

After completing this dissection from the right side of the table, the surgeon shifts to the opposite side to complete the procedure. A special flexible probe with a small, smooth, round tip* (Fig. 5) is shaped into a suitable curve

*Made by George P. Pilling and Son Co., Philadelphia, Pa.

and passed into the interatrial septum immediately alongside the aorta 1 to 2 cm. posterior to the right coronary artery (Fig. 6). To accomplish this the epicardium is first teased away from its reflection onto the aorta just sufficient

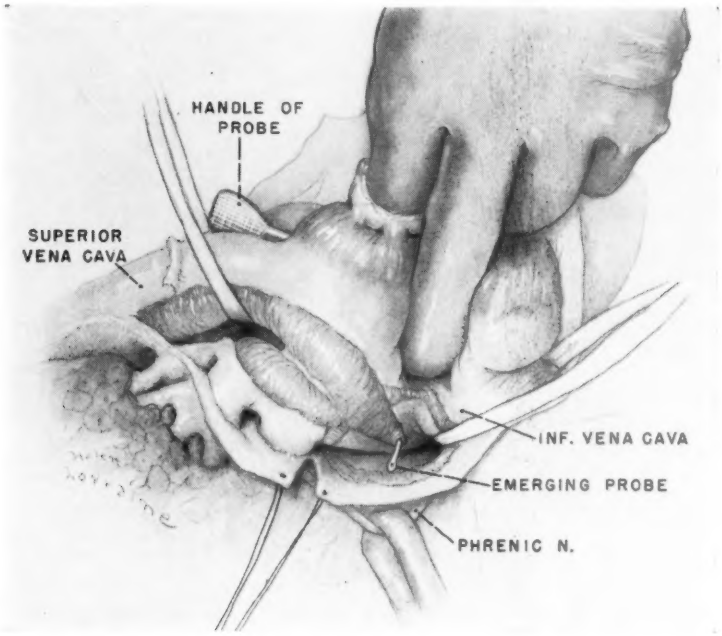

Fig. 7.—The emerging probe is seen as it passes around a low septal defect.

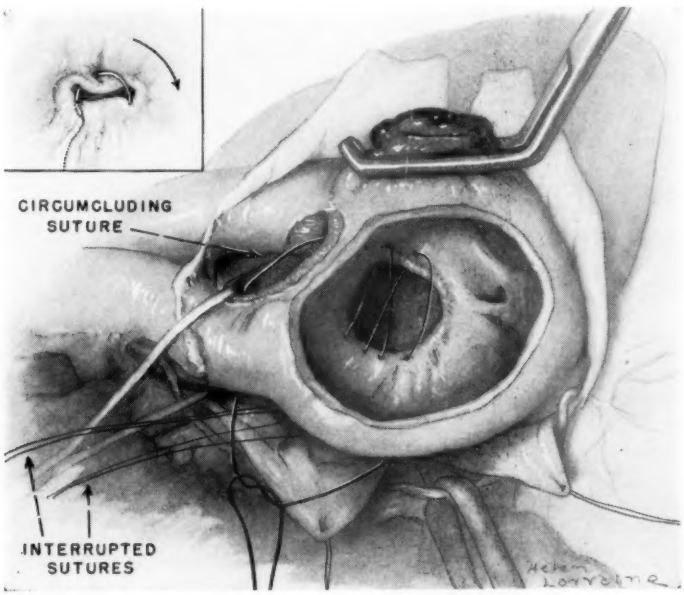

Fig. 8.—The circumcluding suture has been passed. Temporary contriction facilitates the strategic placement of two interrupted sutures. (See inset.) Recently three sutures have been employed.

to admit the probe. Careful digital control with the left index finger is maintained within the atrium. It is important to avoid thrusting the probe into either of the atrial chambers. The probe is allowed to slip easily into

the interatrial septum and around the margin of the defect, which it will do with very gentle guidance. If necessary, the probe is withdrawn, reshaped, and reinserted.

The probe may emerge posteriorly at the inferior margin of the defect, but if it hangs at the posterior auricular myocardium, a small incision is made

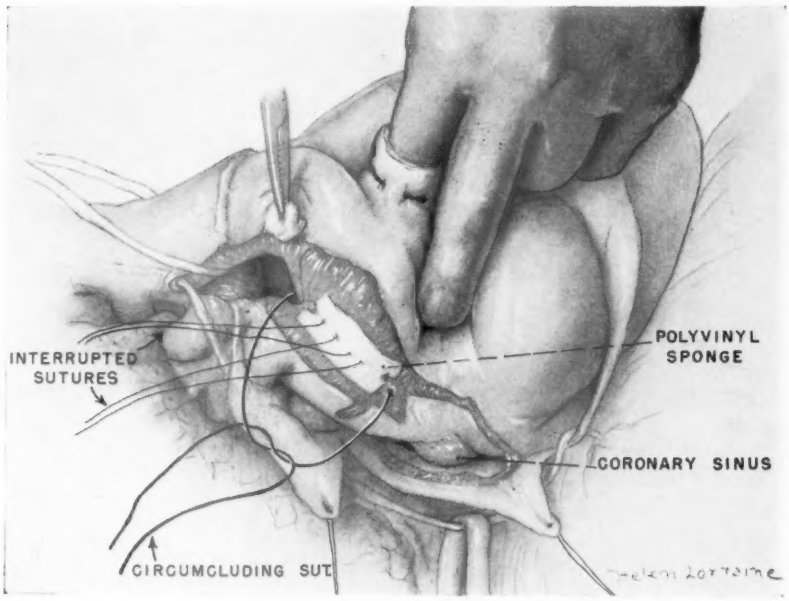

Fig. 9.—Circumclusion is controlled by the intra-atrial palpating finger.

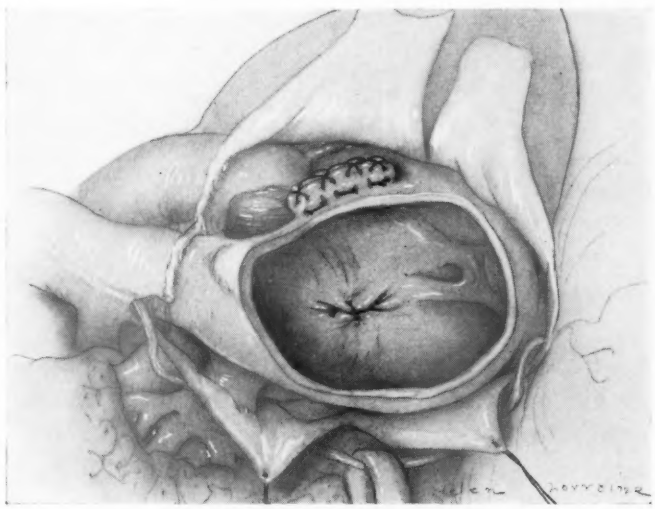

Fig. 10.—A smooth closure with minimal tension has been obtained.

over its tip (Fig. 7). As the probe appears, it is steadied with a needle holder while the surgeon, using his right hand, threads the eye with a No. 1 nonabsorbable suture. The suture is then withdrawn through the septum on the probe and is subsequently passed around behind the superior vena cava. With this circumcluding suture the first assistant temporarily constricts the defect down

to finger-tip size. This permits the surgeon to insert easily two interrupted sutures of No. 00 silk on a small curved needle from the interatrial cleavage posteriorly so as to catch up the anterior and inferior rim of the defect *around the circumcluding suture* (Fig. 8). These sutures are subsequently rethreaded and passed back through a compressed polyvinyl sponge.*

The first assistant ties down the circumcluding suture over compressed polyvinyl sponge and stops the constriction when the surgeon ascertains complete closure by digital control (Fig. 9). The first knot is held with a hemostat as the tie is completed. The two interrupted sutures are then tied and complete closure is assured (Fig. 10). Excess polyvinyl sponge is trimmed away. The knots and polyvinyl sponge will lie completely hidden in the interatrial cleavage plane as the right atrial wall is released.

DISCUSSION OF TECHNIQUE

We have modified slightly the circumclusion technique of Sondergaard by employing several interrupted sutures of 2-0 silk to supplement and strengthen the closure; thus we avoid complete reliance on the single circumcluding suture. The interrupted sutures are easily inserted with a small curved needle even in the case of large defects after temporarily constricting the defect to approximately finger-tip size. These sutures are now employed routinely and are placed most accurately before final complete constriction when finger-tip control is still possible. By passing these sutures around the circumcluding strand in the anterior and inferior rim one eliminates any tendency of the interrupted sutures to cut through. The additional assurance of permanent and complete closure afforded by these simple sutures seems definitely worth while. A central probe patency which could occur if the circumcluding suture loosens slightly is thereby prevented. Cooley[7] has suggested the use of interrupted sutures when the circumferential suture passed according to the Björk-Crafoord technique fails to close the defect completely. In our experience, with the circumcluding suture passed within the septal plane by the probe, the defect constricts easily to a point; the interrupted sutures are used rather to assure permanence of closure. Complete closure can, of course, be easily tested with a probe passed in through the auricular wall.

A special flexible probe constructed with an eye which will accommodate up to No. 2 silk has greatly simplified the attachment and subsequent withdrawal of the suture into the septal plane (Fig. 5).

The technique is applicable to all ostium secundum defects, however large, except when associated with anomalous pulmonary veins and here a slight modification seems desirable. We have been pleasantly surprised at the freedom from serious tension after closure of large defects. However, some other technique must be employed for ostium primum defects because of the complete absence of septal tissue immediately above the atrioventricular valves.

*One centimeter slice compressed to one millimeter, soaked in penicillin and streptomycin solution.

Several closed techniques are widely used including the atrioseptopexy of Bailey,[1] direct suturing methods described by Gross and Watkins,[9] the techniques of Kay[11] and O'Neill,[20] and the method of Lam[13] employing a two-ended needle. The atrioseptopexy of Bailey has several theoretical and practical disadvantages. It is less adaptable to the high and low defects because of the danger of obstructing the caval orifices and the tricuspid valve. The technique distorts the auricular chamber to a considerable degree and does not allow easy detection of small residual openings after the closure. Other direct suture techniques employing multiple sutures are hazardous in the absence of an anterior or inferior rim since the aorta, coronary sinus, or conductive tissue may be endangered. Unless some maneuver is employed to draw the antero-inferior rim posteriorly, an unnecessarily large needle with wide curve will be required. Gerbode[8] has advised the use of a hook inserted through the posterior auricular wall to draw the anterior rim of the defect posteriorly. None of the disadvantages cited applies to the probe technique of circumclusion in which a probe is easily passed even in the absence of an anterior or inferior rim. We have had no difficulty in elevating endocardial tissue with the probe for closure in those cases in which a rim fades out completely in the region of the inferior vena cava. In these latter cases which comprise 25 per cent of our entire series, we appreciate especially the superiority of the probe over the large curved needle for passage of the suture.

The one outstanding hazard which we recognize is the possibility of establishing a false passage into either of the atrial chambers in the corner between the aorta, right atrial wall, and coronary artery at the point where the probe is initially inserted. Although usually of no serious consequence this could and did lead to serious hemorrhage in one instance during subsequent circumclusion and was controlled only after great difficulty because of the very tight angle allowed one for maneuvering a needle. It is important to emphasize, therefore, that the probe should be inserted initially into the proper plane under digital control, a rather simple maneuver. The reflection of the epicardium onto the aorta is dissected away only sufficiently to admit the tip of the probe. Unless care is exercised the probe may also break into the atrial chamber just as it is about to emerge posteriorly, as it did in one of our cases. The small cleft which may remain after constriction is then closed by the most inferior of the interrupted sutures. With experience this break in technique can be avoided.

REVIEW OF CASES

Of the 16 patients in this series the first 4 fall into a separate category since the technique of circumclusion was not then employed. Case 1* presented an ostium primum defect with well-formed endocardial cushions, a most unusual anatomic situation. The defect was closed with four interrupted sutures inserted into the left side of the interventricular septal margin from the posterior auricular wall. This patient has obtained an excellent clinical result with apparent complete closure of his defect. Case 2*, a 45-year-old

*Cases 1 and 2 operated upon with the assistance of Dr. Elton Watkins.

woman in heart failure, was found to have a large central defect with a veil-like rim. A number of interrupted sutures were inserted from the posterior aspect of the auricular wall, but these failed to close the defect completely, and a central opening 1.5 cm. in diameter remained. The procedure was complicated by considerable hemorrhage and ventricular fibrillation. Defibrillation was accomplished after 40 minutes of cardiac compression, and after a stormy postoperative course the patient was discharged from the hospital. Recatheterization showed a reduction in the pulmonary flow to systemic flow ratio from 6 to 1.7. The patient gradually improved for a time, but subsequently died in congestive failure 18 months postoperatively. The third patient, a 3-year-old child, proved to have an atrioventricularis communis with mitral insufficiency. An attempt was made to close the defect using a

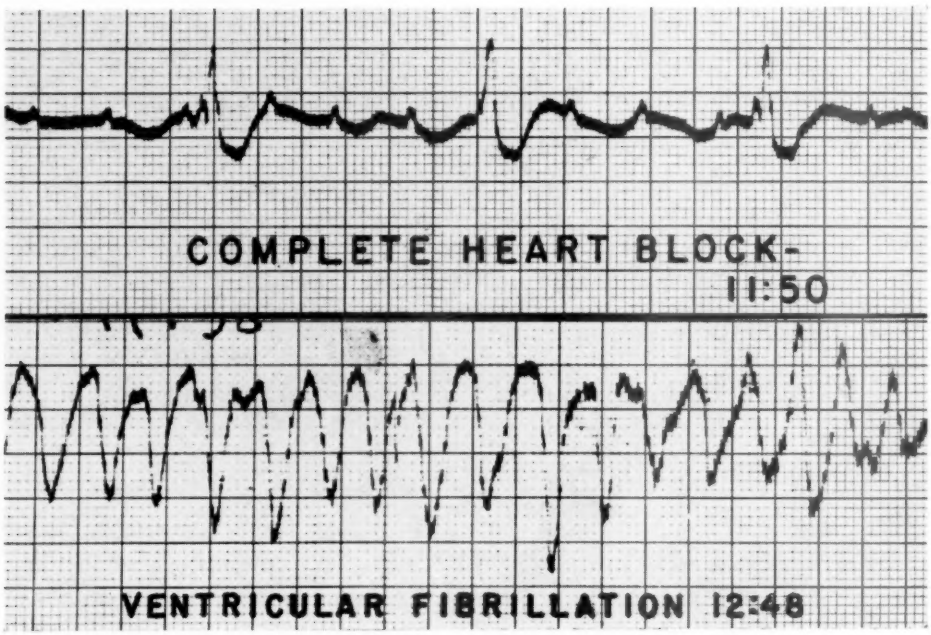

Fig. 11.—Case 3 (Atrioventricularis communis). Attempted passage of large curved needle led to complete heart block followed by ventricular fibrillation and death.

large curved needle according to the technique of Björk and Crafoord. This led to complete heart block which persisted even after removal of the suture and the development shortly of ventricular fibrillation which could not be reverted (Fig. 11). This case was referred to by Dr. Zoll in a recent paper.[24] Case 4 proved, on exploration, to have an ostium primum lesion with no palpable valvular insufficiency and no attempt was made to close the defect. The patient continues at this time in moderate congestive failure and consideration is being given to re-exploration and attempted closure.

Thus, among our first 4 patients we encountered 2 with ostium primum defects, 1 with an atrioventricularis communis, and the fourth with a large defect which was incompletely closed. Our remaining 12 patients have all

presented either ostium secundum defects or high defects associated with anomalous pulmonary venous drainage. A somewhat simplified classification of atrial defects is shown in Table I.

TABLE I. CLASSIFICATION OF DEFECTS

	SMALL*	MODERATE†	LARGE‡
Ostium primum		2	
Atrioventricularis communis		1	
High defects (with anomalous pulmonary venous drainage)		2	
Ostium secundum			
(a) Central (foramen ovale defects)	1	5	1
(b) Central (extending low to inferior cava—no lower margin)		2	2
	1	12	3

*Small—less than 2 cm.
†Moderate—2 to 4 cm.
‡Large—larger than 4 cm.

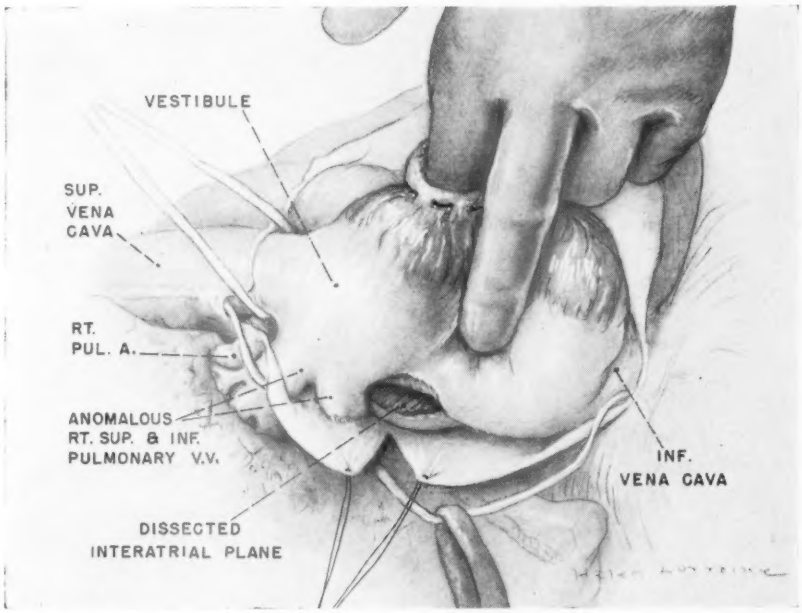

Fig. 12.—Essential anatomy encountered in two cases with defect superior to fossa ovalis and with anomalous pulmonary venous drainage.

We have encountered two high defects associated with anomalous venous drainage of the type accurately described in a recent paper by Lewis[15] (Fig. 12). In both instances a small vestibule was found at the upper end of the atrial chambers into which the superior vena cava and anomalous pulmonary veins drained. The vestibule was common to both right and left atria through a high septal defect which measured 2.5 to 3 cm. in diameter. The defect lay superior to the fossa ovalis which was perfectly formed. The anomalous veins entered the vestibule slightly to the right of the septal plane. In neither case was there a posterior rim to the septal defect. The inferior rim coincided with the inferior margin of the lowest anomalous vein. The interatrial cleavage plane originated at this point and could be developed deeply so that a probe

was easily passed from the posterior aspect through the septum above the fossa ovalis to a point superior to the interatrial muscle bundle. In one of the two cases both the superior and inferior pulmonary veins from the right lung drained into the vestibule. The superior vena cava was small since the patient also had a left superior vena cava draining into the coronary sinus, as determined by catheterization studies. Because of the small size of the right superior vena cava it was possible to utilize the standard circumclusion technique, turning in the pulmonary veins posteriorly through the defect while tying the circumclusion and interrupted sutures anterior to the pulmonary veins. In the other patient the superior and middle lobe veins drained into the vestibule and the inferior pulmonary vein into the left atrium. The superior vena cava was normal in size. This patient also had an azygos "lobe" with venous drainage into the superior vena cava 2 inches above the vestibule.

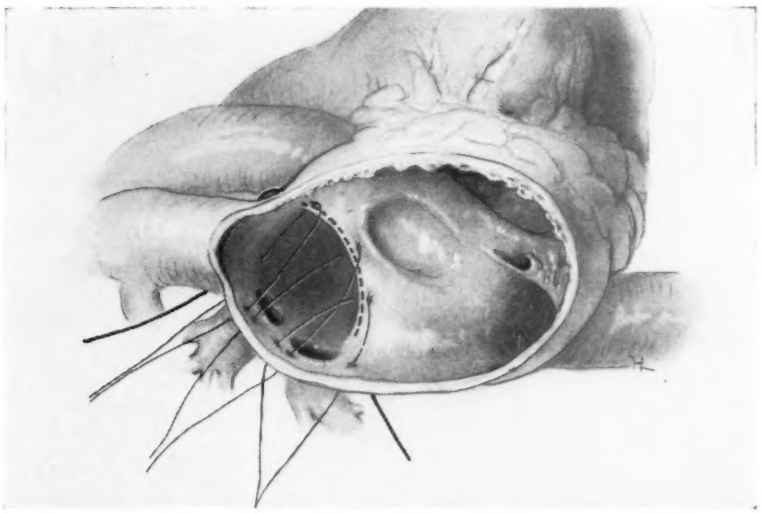

Fig. 13.—Closure in previous instance (see Fig. 12) by interrupted sutures facilitated by circumcluding suture (see text).

The azygos lobe was resected because it did not seem feasible to transplant its small vein. The circumcluding suture was used to draw the anteroinferior rim forward and to facilitate placement of interrupted sutures, but it was not tied down because of the possibility of constricting the orifices of the pulmonary veins. Instead, it was cut off and a segment left in the interatrial cleavage plane. (Figs. 13 and 14.) In a future case we would consider anchoring each end of this segment posteriorly, one end just behind the superior cava and the other at the lowest margin of the defect.

Among the remaining 10 patients we have encountered foramen ovale defects of various sizes and positions, some with a very adequately developed posterior rim and others with a very small posterior rim. In four instances an inferior rim was absent with the defect extending low to the region of the inferior vena cava. As pointed out by Lewis[15] in these cases, the caudal wall of the left atrium merges into the wall of the right atrium at the lower part of the defect. In general, these defects are somewhat larger than the average

foramen ovale defects. In one patient we encountered a small defect 1.5 cm. in length and 0.5 cm. in width, centrally located in the fossa ovalis. Although this defect was successfully closed by the circumclusion technique, undoubtedly some bunching of tissue occurred since the fossa ovalis was only partially defective. As a result, a diverticulum was probably formed in communication with the left atrium. For this type of atrial defect and for multiple small defects it might be more acceptable to make the closures with one or two interrupted sutures through a Gross well. Björk and Crafoord[5] and Watkins and Gross[27] have reported embolic complications occurring from suture material and from tissue eversion into the left atrial chamber. It is important, therefore, that a smooth closure be obtained with a minimum of exposed suture material.

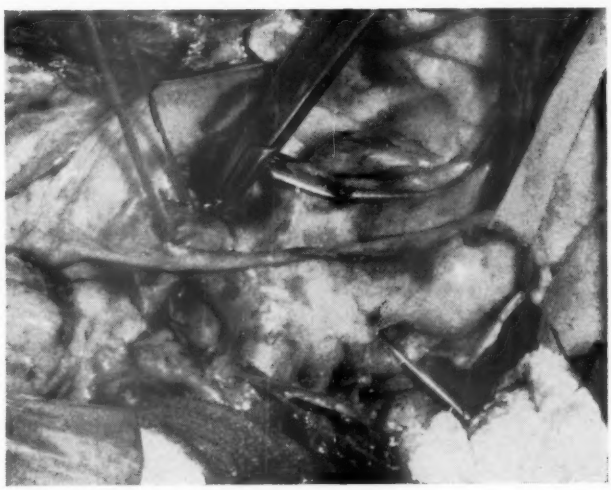

Fig. 14.—Case 13 (see Fig. 12). The operative view shows the probe passing through the septal plane from posterior aspect anteriorly. Tip of probe is steadied with needle holder. Superior and middle lobe veins drain anomalously.

We have recognized no instances of associated mitral stenosis in our series. One patient was found to have a thrill in a somewhat dilated ascending aorta at surgery, and after closure of the septal defect the thrill increased markedly. Prior to surgery we had not suspected aortic stenosis, but had worried about the possibility of an ostium primum defect which was suggested by a high-pitched systolic murmur heard near the apex. No murmur was heard over the aortic area. Postoperatively the aortic systolic murmur was easily audible.

Although mild gradients over the pulmonary valve have been noted in a number of patients, in only one instance has the gradient seemed very significant. In this patient the right ventricular systolic pressure was 64; the pulmonary artery pressure was 20. Pressures were taken after closure of the interatrial septal defect and no gradient could be demonstrated. Postoperative catheterization studies revealed a right ventricular pressure of 20 systolic and a pulmonary artery pressure of 16. Thus, when the degree of pulmonary stenosis appears moderate and the left to right shunt through the interatrial septal defect is large, it would seem advisable to close the

defect first before assuming that pulmonary valvulotomy is necessary. Simultaneous pulmonary valvulotomy and atrial septal defect closure has been reported by Björk and Crafoord[6] and by Kirklin.[12]

Two of our patients have presented other stigmata of Marfan's syndrome with arachnodactyly, high palate, and thoracic skeletal deformities. An additional two patients have exhibited a localized pectus carinatum in the upper sternal region, the so-called arcuate protrusion deformity.

RESULTS

In evaluating our results we are cognizant of the difficulties of establishing complete closure with absolute certainty. Neither oxygen saturation determinations nor blue dye studies are likely to demonstrate conclusively the presence of an interatrial shunt less than 20 per cent of the pulmonary artery flow. The nitrous oxide method[17] of detecting small left to right shunts is attractive, but the analysis is time consuming and we have been able to carry out this study in only a few patients.

We agree with Kirklin[12] and with Swan[26] that nothing short of complete closure should be accepted since even small defects may allow large shunts.

TABLE II

NUMBER	AGE	SYMPTOMS (1 TO 3)	PREOPERATIVE SHUNT RATIO*	POSTOPERATIVE STUDIES			POSTOP. SHUNT	CLINICAL RESULT
				CATHETERIZATION	BLUE DYE	NITROUS OXIDE		
1. (E. G.)	17	1	7.2	+	+	-	0	Good (ostium primum)
2. (R. J.)	45	3	6.0	+	-	-	1.7	Died 18 months postoperative
3. (C. F.)	3	3	2.2				-	Operative death (A-V communis)
4. (B. M.)	8	3	8.5				-	Exploration only (ostium primum)
			Circumclusion Technique Adopted					
5. (W. M.)	23	1	4.1	-	+	-	0	Good
6. (C. H.)	21	2	3.6	+	+	+	0	Good
7. (B. Y.)	11	0	5.6	+	+	+	0	Good
8. (B. B.)	7	1	2.7	+	+	-	0	Good
9. (S. R.)	9	0	2.8	+	+	-	"Probe patency"	Good
10. (B. H.)	28	2	3.2	+	+	+	0	Good
11. (M. H.)	15	1	3.3	+	+	-	0	Good
12. (B. T.)	14	1	6.3	+	+	-	0	Too early
13. (F. R.)	8	1	3.5	-	+	-	0	Too early
14. (L. H.)	4	0	3.5	-	+	-	0	Too early
15. (H. A.)	28	1	2.9	-	+	-	0	Too early
16. (M. H.)	33	3	5.0	-	-	-	-	Too early

*Pulmonary flow to systemic flow ratio.

This fact was impressed upon us by Case 11, a large girl of 15 years with a pulmonary-systemic flow ratio of 3.3. The defect was roughly estimated to measure only 1.5 by 0.5 cm., yet closure of this small defect has completely eliminated the shunt.

The results in the entire series of 16 cases are tabulated in Table II. All of the surviving patients in whom closure was attempted, except one, have been studied either by postoperative cardiac catheterization or by blue dye injections.* No functional shunt can be demonstrated in any of the patients. In two cases the blue dye curves might be considered equivocal; in one of these patients the catheterization findings were convincingly normal; in the other a nitrous oxide study was entirely normal. In one patient with normal postoperative catheterization values and a normal dye curve, the catheter passed through a defect in the septum into a pulmonary vein. This demonstration of "probe patency" has strengthened our conviction regarding the desirability of interrupted sutures to supplement the circumcluding suture.

The clinical improvement in patients previously exhibiting significant symptoms has been gratifying. Exercise tolerance has been strikingly increased; fatigue and exertional dyspnea have been eliminated. A pulmonic murmur persists in only one patient, recently operated upon, and this is very faint. The blue dye curve is entirely normal.

DISCUSSION

Our experience is similar to that of Sondergaard who has now operated upon more than 15 patients by the technique of circumclusion and reports closure (by catheterization) in all.[21] The results in our series are likewise very satisfactory.

Many patients with interatrial septal defects when first considered for surgery are essentially asymptomatic, or the degree of disability is not great. Nevertheless, the hearts of these patients are moderately enlarged and catheterization studies reveal rather large shunts. In some a moderate degree of pulmonary hypertension exists. Some of these patients will continue with only minimal to moderate disability for years. For these patients, especially, a simple and safe as well as effective surgical technique is desirable, if surgery is to be recommended. These criteria appear to be met by the technique of circumclusion, as described in this paper. Admittedly, the procedure is unacceptable for ostium primum defects and atrioventricularis communis, but here the difficulties faced by other techniques are almost equally as great. Circumclusion can be employed for most other atrial defects. Extracorporeal circulation at this time appears unjustified in the surgical treatment of the average atrial defect. Open heart surgery done under hypothermia, while yielding good results in the hands of those experienced with its use, is unlikely to prove as safe for the seriously ill patient.

The indications for surgery of an interatrial septal defect are not yet clarified, but this is partly due to the feeling that the surgery of this condition entails an operation of considerable magnitude. With increasing experience

*Grateful acknowledgment is accorded Dr. David Richardson for his aid with the blue dye studies.

with the technique of circumclusion we now have little hesitation in recommending closure even in the essentially asymptomatic patient if the pulmonary blood flow exceeds the systemic flow by two to one. The desirability of preschool closure for such patients, as in the case of patent ductus, must be given serious consideration.

SUMMARY AND CONCLUSIONS

1. The Sondergaard technique of circumclusion together with certain modifications is described in detail. The safety, simplicity, effectiveness, and wide applicability of this procedure is stressed. The technique is not suitable for ostium primum defects.

2. A series of 16 surgically treated patients with various types of interatrial septal defects is reported. There was one operative death in a patient with atrioventricularis communis and one late death occurring in an older patient with a large defect in whom complete closure was not accomplished.

3. Twelve patients have been operated upon by a technique of modified circumclusion with no mortality and with apparent functional closure in all. In one patient an opening sufficient to admit the cardiac catheter is known to persist.

4. The clinical results in the surviving patients have been most gratifying.

ADDENDUM

Clinical examination in one patient 9 months postoperatively suggests a recurrence. Blue dye studies and cardiac catheterization 4 months postoperatively failed to reveal a shunt. In this patient only one interrupted suture was used. Further studies will be made.

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DISCUSSION

DR. CONRAD LAM (Detroit, Mich.).—I should like to agree with Dr. Bosher that it is unnecessary to apply such techniques as hypothermia in the repair of all interatrial septal defects. Three cases out of four do not need open heart surgery. I have been surprised that it is possible to close a large defect by a purse-string suture. We do not use such a suture to close hernias. Dr. Bosher adds stitches across the partially closed defect.

(Slide.) I should like to give a further testimony for the method we described a year ago, the method with the pointed needle. After a thorough Sondergaard dissection it is quite easy to pass interrupted sutures through with the two-pointed needle. I will repeat again, it seems that it is not good surgical practice to try to close this defect with a circumcluding suture.

We have now closed approximately 35 septum secundum defects by this method without a mortality. Our results with the septum primum defects have been similar to those reported by other groups.

DR. DENTON COOLEY (Houston, Tex.).—I have enjoyed Dr. Bosher's presentation very much and am interested in some of the technical improvements he has made in Sondergaard's method of atrial septal repair. I am convinced from our experience with closed methods of repair of atrial defects that a large number of these defects can be successfully and permanently corrected by circumferential suture. At this time we have operated upon 17 patients by this method with complete closure of the defect in 14, incomplete in 3 with death occurring in one of the unsuccessful closures about 10 days later. Our method consists of circumferential suture with a large needle and heavy silk suture and reinforcement with several mattress sutures.

Unfortunately not all atrial defects are correctable by this relatively simple maneuver, and it may not always be possible to differentiate the lesions before operation. Defects such as the auriculoventricular communis and ostium primum are often impossible to correct by this method and many high defects in the septum, particularly those associated with anomalous pulmonary venous or caval drainage, are difficult if not impossible to correct by transcardiac

suture. For these lesions open operation will usually provide a complete correction with relative technical simplicity. Hypothermia has been widely used for this purpose by Swan, Lewis, and others and we have used it in four cases. Because of limitations on time of caval occlusion under hypothermia and several other objections such as cardiac arrhythmia and ventricular fibrillation, I believe that this method will not have lasting appeal. Actually the methods of extracorporeal circulation are fairly well developed already, and there is no reason to confine application of this method of treatment to ventricular defects. We have used the bubble oxygenator and pump successfully in 12 patients with ventricular defects and intend to use it in the near future in practically all cases of atrial septal defect because it is unquestionably the best method for open heart surgery.

DR. PAUL T. DE CAMP (New Orleans, La.).—I would like to congratulate Dr. Bosher on this report, and I merely want to state that in our small experience with this same technique, we have been quite satisfied with it and feel it is perhaps the best of the closed procedures.

There are one or two technical points that I would like to emphasize. In case you are tired of the new instruments, we have found that a three millimeter Bakes dilator with a hole drilled through the end of it to receive the ligature makes a very satisfactory probe. For those of you who have abdominal convictions, such an instrument will be available.

The other thing that I would like to state is that in our experience the most critical point where one may get into trouble and where you may wish you had better exposure is at the lower end of the tunnel, which is behind and medial to the inferior vena cava. We have found it very helpful to free up and incise the diaphragmatic pericardium posteriorly, just medial to the cava resulting in adequate exposure of this critical area.

DR. JULIAN JOHNSON (Philadelphia, Pa.).—I might say that I had an opportunity to talk to Dr. Sondergaard this summer when we were in Copenhagen. He now operates upon children with atrioseptal defects as soon as the diagnosis is made, even though they are asymptomatic. He feels that they can be done as elective procedures in the same manner as a patent ductus arteriosus. At the time I talked to him he had done eleven by using the probe passed between the atrium with no deaths. I believe these were all in children.

DR. LEWIS H. BOSHER (closing).—I wish to thank all of the discussers for their interesting comments.

I would like to point out that Dr. Sondergaard himself does not use interrupted sutures and he feels that he gets satisfactory closure.

In a recent letter from him I learned that he has done more than 15 cases by this technique and catheterization studies show all of them are closed. I would like, however, to point out that it is rather difficult to detect small shunts by catheterization methods and blue dye studies fail to pick up shunts less than 15 per cent of the pulmonary flow. I will remind you that in one instance the cardiac catheter passed through a small opening in the septum, although the cardiac catheterization and blue dye studies indicated no shunt.

(Slide.) There were two cases encountered in which variations of the technique were utilized. These cases had anomalous pulmonary venous drainage with high defects located superior to the fossa ovalis. This is a drawing of such a case. This is a small vestibule located superiorly with the pulmonary venous drainage into the vestibule.

You will note that the interatrial cleavage does not extend upward all the way, but terminates at this point. The probe was passed from the posterior to the anterior side, actually superior to the interatrial muscle bundle. Here is the probe entering here. Here it comes out. Here is the vestibule. In one of the cases the inferior pulmonary vein entered the left atrium; in the other, both right pulmonary veins entered the vestibule.

(Slide.) The next slide shows how we dealt with the superior vena cava. Because the patient had a left superior cava, and the right cava was small, one could circumclude without constricting the pulmonary veins. In the other case the superior cava was large and we cut off the circumferential suture without tying it and left it in the cleavage plane.

In answer to Dr. De Camp's question about the point at which the probe emerged in the drawing, I certainly agree with him, that in most of the cases the probe comes out somewhat lower.

THE SURGICAL TREATMENT OF PERSISTENT COMMON ATRIOVENTRICULAR CANAL

JACK C. COOLEY, M.D.,* JOHN W. KIRKLIN, M.D.,** AND HARRY G. HARSHBARGER, M.D.,*** ROCHESTER, MINN.

PERSISTENT common atrioventricular canal is a congenital malformation of the heart which combines the complexities of defects of the atrial and ventricular septa with defects in the atrioventricular valves. Although it was a pathologic curiosity for years, more recently this lesion has taken on new significance from both the diagnostic and surgical standpoints. At present, it poses a therapeutic challenge which can, in many cases, be met with success.

EMBRYOLOGIC ASPECTS

Between the fourth and sixth weeks of life of the human embryo, two endocardial cushions, one dorsal and one ventral, appear at the junction of the single atrium and ventricle, the atrioventricular canal.[8] They grow in the midline of this canal, ultimately coming in contact with each other, and they divide the canal into right and left halves. While these changes are occurring, the primary interatrial septum and muscular interventricular septum are developing. The interatrial septum grows down from the superior and dorsal walls of the atrium, narrowing the communication between the two atria, the interatrial foramen primum. Similarly, the muscular portion of the interventricular septum grows upward from its anlage at the ventricular apex to narrow the communication between the two ventricles. When the endocardial cushions have united anteroposteriorly, they send out a superior prolongation which meets the developing atrial septum and an inferior prolongation which joins the ventricular septum. The anlagen of the anterior halves of the anterior leaflet of the mitral valve and the septal leaflet of the tricuspid valve lie in the ventral atrioventricular endocardial cushion, while the anlagen of the posterior halves of these cusps lie in the dorsal cushion. The result of failure of proper development and fusion of these endocardial cushions produces a defect in the lower atrial septum, a defect in the upper posterior ventricular septum, and defects in the anterior leaflet of the mitral valve and the septal leaflet of the tricuspid valve. This comprises the lesion known as "persistent common atrioventricular canal."

SURGICAL ANATOMIC ASPECTS

Atrial septal defects, so far as their surgical implications are concerned, can be classified into two groups. The common group is that which might be termed "ordinary atrial septal defect." Such defects are characterized by the fact that the anterior inferior margin of the defect is formed by a remnant of atrial septal tissue. Defects of this type vary greatly in size and location; they may be located virtually anywhere in the atrial septum. They may extend down very near the atrioventricular valves. They are not associated with

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clefts in the atrioventricular valves, although valvular insufficiency can occur. Of 80 patients with this type of atrial septal defect treated surgically by us, seven had mitral insufficiency of such degree as to be palpable at operation.

The second group of atrial septal defects are those in the lower portion of the atrial septum in which there is no septal tissue between the defect and the atrioventricular valve rings. As Edwards has indicated, these can be considered a form of persistent common atrioventricular canal. Some have preferred to term these "ostium-primum defects"; others regard them as "endocardial cushion defects." In this group, defects of the atrioventricular valves occur and valvular insufficiency often is a problem. A cleft in the mitral valve always has been found, in our experience, and the presence or absence of a cleft in the tricuspid valve helps subdivide persistent common atrioventricular canal into two categories: those in which there is a cleft in both valves (the complete form), and those in which there is a cleft only in the mitral valve (the partial form). Each of these forms may or may not be associated with valvular insufficiency.

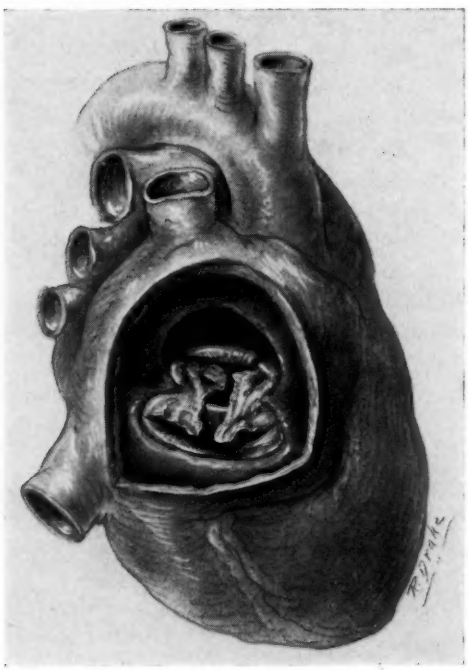

Fig. 1.

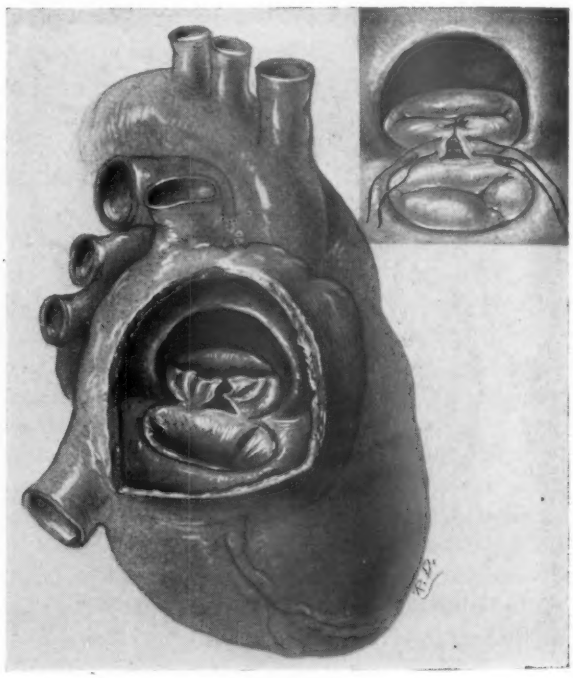

Fig. 2.

Fig. 1.—The complete form of persistent common atrioventricular canal. The defect in the atrial and ventricular septa can be seen. Because of the clefts in both atrioventricular valves, a bare area of ventricular septum is seen forming the inferior margin of the defect.

Fig. 2.—The partial form of persistent common atrioventricular canal. There is a cleft in only the mitral valve. The tricuspid valve is intact across the atrioventricular valve ring. The insert demonstrates the technique used for repair of a defect in the mitral valve when mitral insufficiency is significant.

In the complete form of persistent common atrioventricular canal (Fig. 1), there are clefts in the anterior leaflet of the mitral valve and in the septal leaflet of the tricuspid valve. Since there is no valvular tissue across the midportion of the atrioventricular valve ring, a bare area of ventricular septum is seen forming the central portion of the lower margin of the defect.

The partial form differs in that there is a cleft in only one of the atrioventricular valves, the anterior leaflet of the mitral valve (Fig. 2). Because there is tricuspid valvular tissue intact across the atrioventricular valve ring, no bare area of ventricular septum can be seen. An interventricular communication may occur below the valve leaflets in either form.

Knowledge of the exact anatomic location of the bundle of His in the presence of persistent common atrioventricular canal has long been needed, for an inadvertently placed stitch through or around this bundle may lead to complete heart block with fatal termination. Recently Lev,[6] using tissue-dissection technique and serial sectioning, has shown that when persistent common atrioventricular canal is present the bundle of His courses along the posteroinferior margin of the defect. Because of this, sutures placed in this margin must not include large amounts of tissue and should not be continuous.

SURGICAL INDICATIONS

When the diagnosis of persistent common atrioventricular canal has been established, operation should be advised, in our opinion, unless a specific contraindication is present. Without surgical correction, the outlook for a normal life span is indeed doubtful. The majority of these patients are symptomatic when the lesion is first discovered.

There is one complication of persistent common atrioventricular canal which, when identified, leads us at present not to recommend operation: that is, increased pulmonary resistance of sufficient severity to cause the predominant shunt to be from right to left. This contraindication is not always easy to identify with assurance, and it probably is diagnosed best by cardiac catheterization.

Under these circumstances, frank cyanosis may be present clinically, and the roentgenologic picture often is that of a relatively small heart. The cardiac shadow may have decreased in size when compared in previous roentgenograms of the same patient when the pulmonary hypertension was less severe. Studies in which the earpiece oximeter is used and made while the patient is at rest, while he is breathing 100 per cent oxygen, and while he is exercising, may provide additional data in support of the status of the peripheral oxygen saturation of the patient.

SURGICAL TECHNIQUE

Closed techniques for the repair of atrial septal defect, such as the atrioseptopexy of Bailey[1] and the encircling suture of Søndergaard,[6] have not been successful in the repair of this defect. Open cardiotomy with the use of hypothermia and inflow stasis has not permitted sufficient time for accurate closure and has been accompanied by a high mortality risk in this malformation.[7, 10] Semiopen cardiotomy in which the atrial well technique of Gross[3] is used allows tactile, although not visual, inspection of the defect and has no time limitation. Despite the difficulty in placing the sutures in the lowermost portion of the defect, repair of common atrioventricular canal can and has been achieved by this technique.

The most satisfactory surgical approach to this lesion at present appears to be open cardiotomy with the patient supported by the extracorporeal circulation of blood through a pump-oxygenator. Currently at the Mayo Clinic, this technique is used in cases in which the diagnosis of common atrioventricular canal has been made or is suspected. Bilateral anterior thoracotomy through the fourth interspace and transection of the sternum are done. With the circulation intact, the interior of the right atrium is digitally explored through

the right auricular appendage, and careful search is made for regurgitation through either side of the common atrioventricular valve. At present if severe regurgitation is noted, repair would nevertheless be done, with attention being directed to the valves as well as the defect at the time of repair. Extracorporeal circulation is then established by means of a Mayo Clinic modification of the Gibbon-type pump-oxygenator.[8]

A long vertical incision is made in the right atrium, and the intracardiac sucker is placed in the right atrium to return intracardiac blood to the pump-oxygenator. The common atrioventricular canal is carefully inspected. The valve leaflets are lifted in search of an underlying interventricular communication, which must be obliterated in the repair. Closure is made by suturing a piece of noncompressed Ivalon sponge (4 mm. thick) of proper size and shape into the defect (Fig. 3).[2] Interrupted sutures of 3-0 silk are employed. The first stitch usually is placed through the bare area of ventricular septum, and the sponge is anchored there. Work is then carried from here forward

Fig. 3.—Schematic sketch of repair of a persistent common atrioventricular canal in which noncompressed Ivalon sponge 4 mm. thick is used. Interrupted sutures of 3-0 silk are used in the repair.

and backward to the junction of the atrial septum with the atrioventricular valve rings, and interrupted stitches are placed through the common leaflet in such a manner as to include the underlying septum and are then carried through the sponge. When the sponge is being sutured to the posteroinferior margin of the defect, the sutures are placed in such a way that tight encirclement of the free edge of the ventricular septal remnant which may contain the bundle of His is avoided.

If significant mitral insufficiency is present, repair of the cleft in the anterior leaflet of the mitral valve is done. The simplest method is direct

closure of the cleft by sutures, the sutures being of interrupted 5-0 silk, as shown in Fig. 2. This method has been used successfully in two clinical cases. It presupposes, however, that the cleft is small and that its edges can be drawn together easily without tension. If the cleft is large, use of a prosthesis may be preferable. In such a case, a paper-thin piece of compressed Ivalon sponge can be fashioned to the size of the defect and sewed in place with interrupted sutures of 5-0 silk.

The incision in the right atrium is closed by two rows of continuous silk sutures and the heart is allowed again to take over the circulation. Decannulation and closure are carried out in the usual manner.

SUMMARY OF CASES

Nine patients who had persistent common atrioventricular canal have been operated upon at the Mayo Clinic. In the first three operations, the atrial well technique was used. The last six were operated upon since the development of extracorporeal circulation by means of a mechanical pump-oxygenator, and this technique was employed for them. In all nine cases a prosthesis of noncompressed Ivalon sponge, approximately 4 mm. thick was utilized to close the defect. There were four men and five women in the group, and the ages varied from 10 months to 32 years, five patients being 20 years of age or older. All patients had symptoms. The number and severity varied from those of a 10-month-old child desperately ill to those of a 20-year-old man who experienced only mild exertional dyspnea and occasional bouts of paroxysmal tachycardia.

All nine patients underwent cardiac catheterization preoperatively. Each patient had significant arterialization in the right atrium as compared to the oxygen saturation of the mixed venous blood. Seven of the nine patients had additional arterialization in the right ventricle. Only two patients had significant pulmonary hypertension, and in both of these the predominant shunt was from left to right. All patients had pulmonary flows higher than the systemic flow, and in each case a sizable left-to-right shunt was present. A right-to-left shunt was found in three patients by means of dye curves only, and in two cases the shunt was calculated on the basis of saturation data. In the table are summarized the intracardiac shunts in eight cases. Concerning one additional patient catheterized elsewhere, data were not available.

TABLE I. DATA ON INTRACARDIAC SHUNTS OBTAINED AT CARDIAC CATHETERIZATION IN 8 CASES OF COMMON ATRIOVENTRICULAR CANAL

PATIENT	SHUNT			FLOWS ON AIR (LENGTH 1 MIN.)	
	LEFT TO RIGHT		RIGHT TO LEFT (PER CENT)	SYSTEMIC	PULMONARY
	AIR (PER CENT)	OXYGEN (PER CENT)			
1	68	--	3 (dye curves)	4.4	13.8
2	35	52	----	5.8	8.9
3	70	--	----	4.1	14.6
4	51	--	5 to 10 (dye curves)	--	--
5	69	65	Small (dye curves)	7.8	20.5
6	52	62	30 (air)	2.0	6.8
7	66	75	----	6.3	20.1
8	80	76	20 on air 24 on oxygen	5.2	19.2

In four of the nine patients mitral insufficiency was demonstrated at operation, and in two it was considered severe. In one of these instances direct-suture repair of the cleft in the mitral valve was successfully carried out. In another case in which operation was done subsequent to this series, suture of the mitral valve also was carried out.

Seven patients survived operation for closure of a persistent common atrioventricular canal. Five of these had not yet undergone cardiac catheterization postoperatively at the

time of this report, although in four a dye curve was prepared at the end of the operative procedure and was found to be normal. In another patient complete closure of the defect was achieved as proved by cardiac catheterization 6 months after the operation.[4] In one patient for whom the atrial well technique was used cardiac catheterization done 7 months postoperatively revealed a residual left-to-right shunt of 35 per cent at ventricular level. Preoperatively, the over-all shunt had been 76 per cent. This patient seemed clinically improved.

There were two deaths in this group of nine patients, both patients being children 10 months old. One of these children died 12 hours postoperatively. Severe mitral insufficiency was identified at operation before repair. The persistent atrioventricular canal was repaired, but nothing was done to the cleft in the mitral valve. The evidence is good that this child's death resulted from unrelieved mitral insufficiency. Currently, such a cleft in the mitral valve would be repaired. The other death occurred early in our experience with extracorporeal circulation and probably was caused by unsuspected hypovolemia and heart block.[5]

SUMMARY

Persistent common atrioventricular canal presents a grave problem to the person thus afflicted. Fortunately, progress has been made in both the diagnosis and the surgical treatment. Although problems in surgical therapy remain, there is reason for encouragement in the fact that repair has been accomplished for nine patients, seven of whom are surviving and have been benefited by operation at the time of this report.

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CONSIDERATIONS IN THE ACCURACY AND SAFETY OF ARTERIOGRAPHY

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THE application of the arteriographic technique to clinical problems by the Portuguese pioneers, Moniz[8] and Dos Santos,[7] ushered in a new era in diagnosis without which many of the advances in modern vascular surgery would have been impossible. Following the presentation by Egas Moniz[8] (1927) of the initial studies on cerebral angiography, the urologist Dos Santos[7] presented 300 cases (1929) in which translumbar aortography was employed. Continuing in the development of abdominal aortography, Castellanos[4] (1937) and later Farinas[11] (1941) described the transfemoral route using a catheter. After a period of hesitation* in this country, aortography was finally made popular through the efforts of Nelson[20] and Doss[6] (1942) working independently. Concomitantly peripheral arteriography developed. Currently, roentgen visualization by radiopaque dye injection of virtually any major artery in the body is a feasible and an acceptable procedure.

Well established as these techniques may be, arteriography is not entirely without hazard; though infrequent, there are numerous complications and defects in accuracy which may occur. It is the purpose of this paper to discuss these aberrations and to suggest ways of avoiding them or of reducing them to a more acceptable minimum. We believe certain criteria can be established for greater safety and greater accuracy in arteriography. These criteria concern the indications and the choice of site of injection, anesthesia, and technique.

Perhaps the most important consideration in the prevention of complications in arteriography is the careful selection of only those patients who possess strictly valid indications for the procedure.

It should be emphasized that it is not always necessary nor even desirable to employ arteriography in arterial disease since many problems in vascular diagnosis can be solved by simpler, safer, and less time-consuming methods. Furthermore, though the incidence of complications is not great, some risk is

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*In 1936 Henline and Moore[15] reported their study on 19 dogs subjected to aortography by the posterior route. Five animals died from hemorrhage and 3 from drug toxicity soon after the injection. As a result of this experiment these workers cautioned against the utilization of aortography, not realizing that hematoma formation and iodine sensitivity are far greater risks in the dog than in man. The consequence was apathy toward the application of aortography to patients in this country for several years.

inevitably present, and when trouble does occur it can sometimes be tragic. The principle of using arteriography sparingly has been thoroughly discussed in a recent editorial by Shumacker.[23]

In symptomatic arterial obliterative disease angiography undoubtedly is of great help in determining the presence and extent of segmental occlusion, and consequently it provides significant information pertinent to the feasibility and mode of treatment. Visualization of the circulation distal to an occlusion is as important as visualization of the occlusion itself. In this respect arterial angiography usually provides more information about the nature of the vascular tree than does surgical exploration alone. However, in instances of borderline circulation, such as those with impending gangrene, ischemic ulcer, and similar changes, arteriography would appear to be contraindicated. In such limbs, the dye-induced spasm may be more than the tenuous circulation can tolerate, and hence the risk of precipitating gangrene is high. Preparatory sympathectomy may lessen this risk.

Ordinarily, the presence of normal bilateral ankle artery pulsations and/or bilateral popliteal artery pulsations makes arteriography unnecessary. However, when pulsations at the popliteal level or lower are greatly diminished in a symptomatic limb, arteriography may yield valuable information upon which a surgical approach may be based.

Aortography is unnecessary in many abdominal aortic aneurysms. It is not a preoperative requisite in every case to know the exact relationship of the renal arteries to the aneurysm since these arteries are usually proximal to it. Even when both renal arteries are involved in the aneurysm, the aortogram may not always show this, depending upon the arrangement of thrombus lining the aneurysm and upon the adequacy of the visualization. Furthermore, both translumbar and transfemoral (retrograde) approaches to aortography are considered somewhat hazardous in the presence of abdominal aneurysm because of such risks as tearing the aneurysm wall, inducing rupture or dissection, or dislodging a portion of laminated clot contained within the aneurysm.

Arteriography is also superfluous in the localization of peripheral emboli. The simpler methods of physical diagnosis supplemented perhaps by oscillometry are safer and less cumbersome.

Regarding choice of the proper avenue of approach, we believe that the observance of some simple rules can serve to minimize the risk of complications. Some may appear too elementary to relate here, but are best mentioned for completeness' sake.

The relative contraindications to the translumbar approach are marked calcification in the aortic wall at the proposed site of injection, aneurysm at the injection level, inability of the patient to tolerate the prone position, distortion of landmarks due to spinal deformity or other factors, and neurologic disease involving the lower extremities. Routine x-rays of the lower thoracic and lumbar spine (anteroposterior and lateral) are required in order to determine some of these relative contraindications and should always be obtained before aortography is done. When a contraindication exists, the transfemoral (or rarely the transbrachial) route may be used if visualization is considered necessary.

A contraindication to the transfemoral procedure is the presence of bilaterally absent or bilaterally abnormal femoral artery pulsations. If the femoral pulse is reduced, suggesting local stenosis, or if it is exaggerated, suggesting local aneurysmal dilatation, the artery should not be punctured at this site. Thus, if the femoral artery is to be used for either retrograde catheterization or local dye instillation, it must exhibit a relatively normal pulse. If pulsations are normal bilaterally, the femoral artery of the limb with lesser symptoms should be used as the channel for retrograde aortography.

Since chronic obliterative arterial disease is a generalized disease, the vessels of both lower extremities should always be visualized despite unilateral symptoms. Furthermore, injection of the femoral artery on the side opposite the lesion spares the more diseased extremity from eventual scarring which may interfere with direct surgery should it become necessary. In arteriovenous fistula and in peripheral aneurysm, it is more permissible to do unilateral arteriography, for these conditions are generally unilateral and the vessel at site of puncture is usually more normal than in arterial obliterative disease.

Thus, in a large category of cases either the transaortic or transfemoral approach may be used with equal safety. In another category are those in which one avenue has definite advantages over the other, while in a few cases both avenues are closed. The last-mentioned group includes patients having excessive aortic calcification as well as absent femoral pulses. In some of these cases it may be necessary to proceed with surgery without aortography, or to attempt visualization by another approach, such as the brachial artery.

The percutaneous injection of the femoral artery should be reserved for thin individuals with an easily palpated artery covered by a minimum of subcutaneous fat. Moreover, the percutaneous method should be abandoned in favor of a cut-down after two or three reasonable attempts at arterial puncture have failed. In general, we believe the cut-down procedure to be the safer of the two methods in femoral arteriography since adequate exposure of the vessel facilitates less needle trauma to the intima, allows better selection of a puncture site in the vessel wall, minimizes hematoma formation, and permits better judgment as to the amount and duration of external pressure after withdrawal of the catheter.

PREPARATION OF THE PATIENT

Preparatory to the actual performance of arteriography certain preliminary considerations are paramount. The criteria previously discussed are considered and the choice of injection site and of anesthesia are made. The patient is informed of the nature of the procedure and is required to sign an operation permit. He is questioned concerning the possibility of iodine sensitivity or previous untoward reaction to the injecton of contrast media. He is informed fully of the nature of the test and its value in the eventual treatment. Though he need not be frightened by undue emphasis on the risks involved, he should know the responsibility is shared by the surgeon and himself.

Approximately one-half to one hour prior to the procedure, premedication is given by subcutaneous or intramuscular injection. The previous meal

and all subsequent intake by mouth have been withheld. The premedication consists of appropriate doses of Demerol (usually 50 to 75 mg.) and pentobarbital sodium (usually 100 mg.). If general anesthesia is contemplated, atropine (0.4 mg.) is added to the premedication. At least 10 minutes prior to the procedure, a test dose (1 c.c.) of diluted contrast medium is administered intravenously. If signs of sensitivity develop, the study is discontinued. Adrenalin and oxygen are always available in the room in case of emergency need.

ANESTHESIA

It is important when using local anesthesia in arteriography to enlist the complete cooperation and understanding of the patient. When this is impossible, spinal or general anesthesia is to be preferred. It is necessary that the patient who is given local anesthesia during arteriography be fully informed in advance of the nature and extent of the transient pain he is likely to experience with injection of dye. He must be instructed not to move during this period, lest the x-ray picture be distorted. Of greater importance, any motion occurring during the translumbar procedure following the insertion of the needle could conceivably result in a tear of the aorta or in abrasive damage to one of its branches. The vast majority of patients are willing and able to conform in this respect and seem to endure the evanescent discomfort quite well, provided the surprise element has been removed by preliminary explanation.

The severe spasm seen occasionally when the procedure is done under local anesthesia may lead to erroneous interpretation of the roentgenogram, as will be described below.

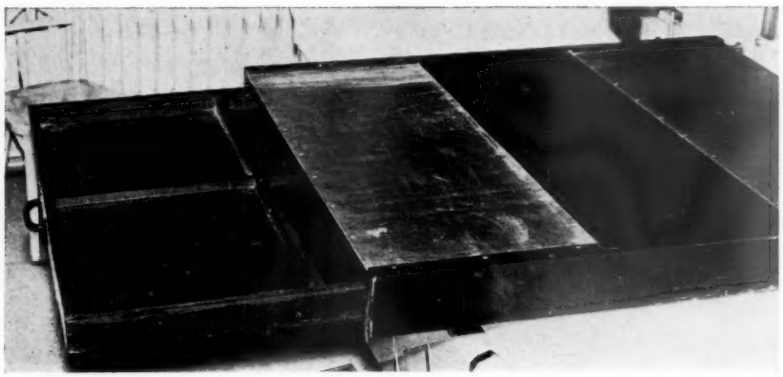


Fig. 1.—Cassette changer, designed to facilitate two consecutive roentgenographic exposures extending from abodominal aorta to mid-calf region. Adapted from Smith and Campbell.[25]

TECHNIQUE

Actual step-by-step technique can be found elsewhere.[24] We will limit our discussion to the technical features which we believe influence the accuracy and safety of the procedure.

For visualization of the lower extremities and abdominal aorta the patient is placed on a cassette changer similar to that devised by Smith and Campbell[25] (Fig. 1). This changer accommodates four 14 by 17 inch grid

cassettes and allows two successive x-ray exposures to be made during a single injection of contrast medium. The area visualized usually extends from the level of L_1 or L_2 down to the lower one-third of the legs. Each of the two exposures lasts one second. They are separated by an interval of three to four seconds, the first being taken during the injection of the last 5 c.c. of dye. The cassette changer is hand-operated during the short interval between the two exposures. The first exposure usually provides good visualization of the abdominal aorta while the second often better demonstrates the peripheral branches in the extremities.

Our technique of translumbar aortography is essentially that described by Smith, Rush, and Evans[24] with minor variations. The aortic puncture is accomplished with a single 6 inch 18 gauge needle with stylet. Although we are aware that in some quarters the 2-needle technique is preferred, we have not found it advantageous. Furthermore, we believe it may conceivably entail unnecessary duplication of certain risks.

It has been our practice to insert the needle 4 fingerbreadths to the left of the midline at the level of L_2-L_3. This approach to the aorta is relatively safe in that it avoids the area between T-12 and L-2 cautioned against by Dos Santos,[7] since the aorta in this area gives rise to the renal and superior mesenteric arteries. Also, choice of this level decreases the likelihood of perfusion of the spinal arteries (via the lumbar arteries) which supply the spinal cord, and it may conceivably minimize the risk of paraplegia as will be discussed below.

Following puncture of the aorta the needle is attached to a fluid-tight system containing sodium citrate solution (2.5 per cent) and consisting of a 12 inch length of polyethylene hose connected to a 20 c.c. syringe. The polyethylene hose connector provides flexibility to the system and yet withstands much higher intraluminal pressures than does rubber tubing. It also allows visualization of the retrograde flow of blood in the system. When it is time to inject the dye, a 30 c.c. Luer Lok syringe containing 70 per cent Diodrast is substituted for the syringe containing the citrate solution. If there is doubt as to whether the point of the needle is within the aortic lumen, a preliminary scout film may be taken following the injection of 5 c.c. of dye. If no extravasation is thus demonstrated, the aortographic visualization may be performed by the injection of 25 to 30 c.c. of 70 per cent Diodrast and the two successive roentgen exposures made as previously described. In order not to exert excessive pressure, we make the injection by hand. It is quite possible to inject the 25 to 30 c.c. of contrast medium within three or four seconds by the hand-operated technique. If the timing of the exposure is accurate, more rapid injection is unnecessary. It is our opinion that overly rapid injection by mechanical devices may predispose to complications. We have found that less dye and less injection pressure are required if proper timing of exposure is employed.

In performing angiographic visualization via the femoral artery, the most important safety feature, in our estimation, is the absence of disease at the site of injection. Gentleness and precision are of prime importance. If unilateral

arteriography is planned, 15 to 20 c.c. of 35 per cent Diodrast is used. For retrograde catheterization and bilateral visualization, 25 to 30 c.c. of 70 per cent Diodrast is injected.

DISCUSSION OF COMPLICATIONS

Thrombosis at Site of Puncture.—This complication may be catastrophic (loss of extremity due to gangrene, infarction of bowel, kidney failure, paraplegia, etc.). Its occurrence may be enhanced by local spasm, particularly when the lumen at the site of puncture is narrowed by intimal disease or when the run-off below this site is poor. Multiple needle punctures or forceful persistence in the presence of difficulty in passing the catheter can undoubtedly produce intimal irritation which may contribute to this complication. Excessive hemostatic pressure on the artery following removal of the catheter may also encourage thrombosis. Demonstration of the patency of an arterial graft by injection of contrast medium—usually an unnecessary procedure—has been known to result in rapid thrombosis of an otherwise patent graft.[13] Thrombosis following arteriography may necessitate emergency surgery.

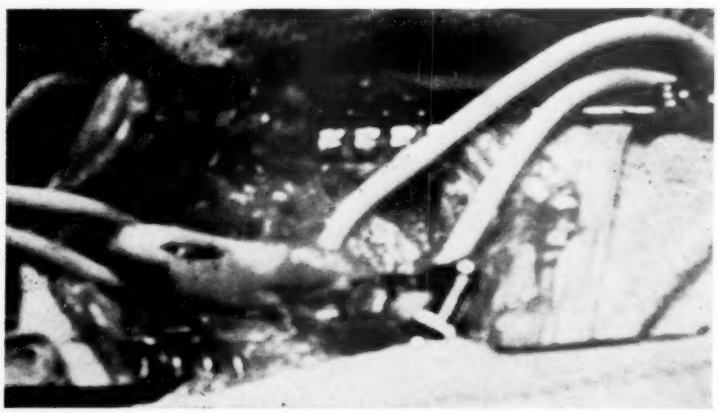


Fig. 2.—Surgical exposure of common femoral artery demonstrating thrombus protruding through arteriotomy site completely occluding vessel at level of previous needle puncture.

One of our patients developed numbness, pallor, muscle weakness, loss of peripheral pulses, and drop in temperature in the right lower extremity during the 24-hour period following an unsuccessful attempt to perform retrograde angiography by way of the right femoral artery. The attempt had failed because the polyethylene catheter had encountered resistance (presumably from an arteriosclerotic plaque) and could not be threaded upward into the aorta. Emergency surgery revealed a localized thrombus at the site of puncture involving the common femoral artery at its bifurcation into the deep and superficial arteries (Fig. 2). Removal of this clot was supplemented with the emergency installation of an arterial homograft. The graft was placed between the common femoral and the upper popliteal arteries in order to bypass a very sclerotic and nearly occluded superficial femoral artery. Following surgery the posterior tibial ankle pulse returned and has persisted now for 6 months, and the limb is free of symptoms.

Another patient in whom a transfemoral approach was used to visualize a segmental occlusion of the same vessel was later subjected to insertion of a by-pass homograft. The operation was performed several days following arteriography, but at surgery a thrombus was discovered at the needle puncture site in the common femoral artery proximal to the anastomosis. This thrombus was removed, but the ankle pulsations suddenly disappeared 6 hours postoperatively. This suggested re-formation of the thrombus occluding an opening to the by-pass.

We observed mesenteric thrombosis in a single case following multiple translumbar aortic injections of the contrast medium in one of our early patients with Leriche syndrome (Fig. 3). Here, as in the case of renal damage, poor terminal aortic run-off predisposes to the complication. We believe this risk may be minimized by injecting the aorta at a point lower than the origin of the superior mesenteric artery (i.e., below the L_1 and L_2 interspace).

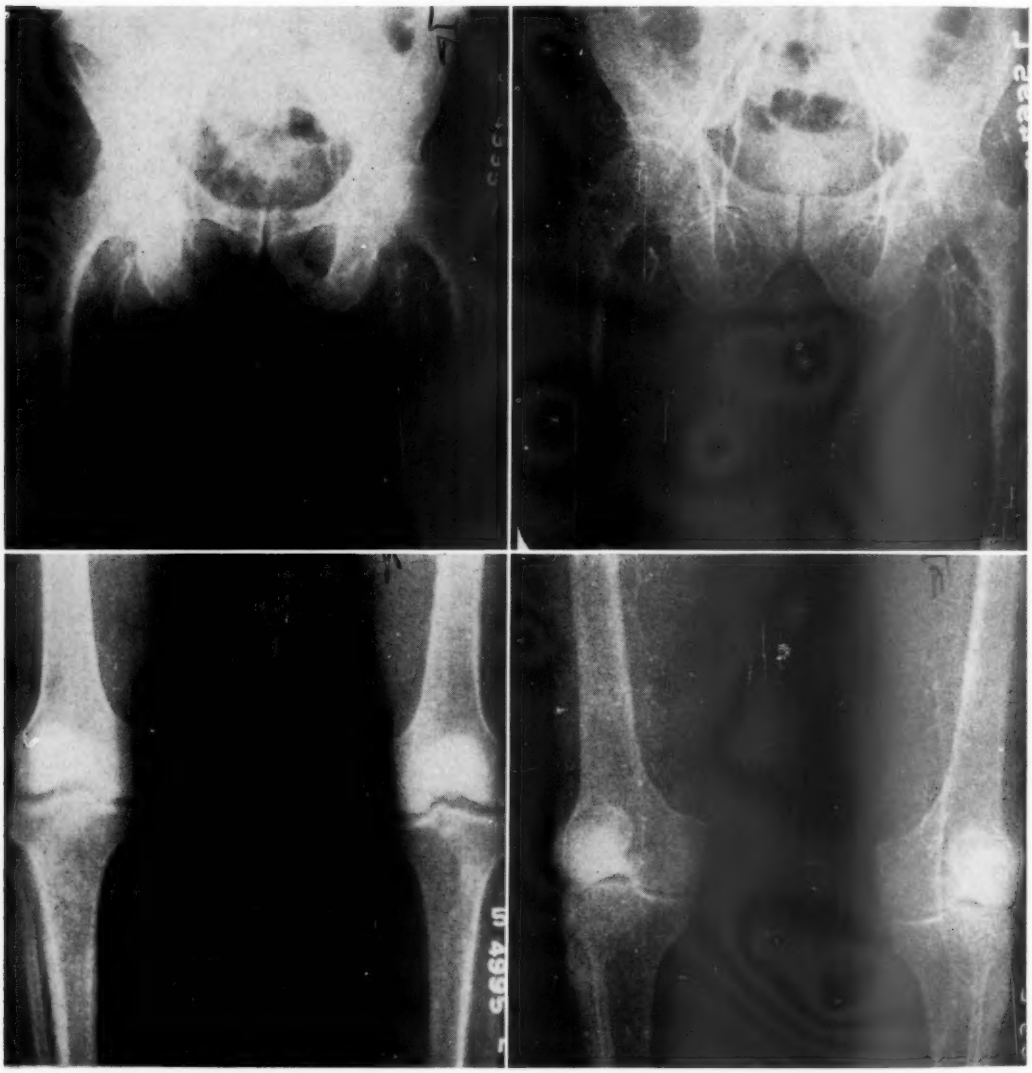
Fig. 3.—Necropsy demonstration of thrombosis of superior mesenteric artery showing gangrene of bowel at lower right.

Hemorrhage or Hematoma Formation at Site of Arterial Puncture.—To a limited extent this occurrence is probably the usual consequence of most percutaneous (including translumbar) procedures. Spontaneous resolution is the rule, and only rarely are serious sequelae observed. However, at least two fatal cases of hemorrhage have been reported following translumbar aortography.[14] In the case of femoral puncture, the judicious use of pressure after withdrawal of the catheter prevents excessive local bleeding.

Transient Arterial Spasm.—Narrowing of the arterial lumen either locally near the point of needle puncture or throughout the injected vascular tree may lead to erroneous interpretation of an arteriogram due to poor radiographic visualization. If such a functional response appears excessive, the arteriographic procedure may be repeated at a later date under spinal or general anesthesia.

In one of our patients translumbar aortography under local anesthesia (using 70 per cent Diodrast) was associated with so much arterial spasm that

no filling occurred below the mid-thigh, and almost no collateral vessels were demonstrated (Fig. 4, *A*). Several days later, however, aortography was repeated, this time under spinal anesthesia via the transfemoral route and using 52 per cent Diodrast. Good distal filling and excellent visualization of collaterals was obtained (Fig. 4, *B*). The second set of pictures revealed a segmental occlusion amenable to surgery, a totally different impression than that obtained originally in which the diagnosis of terminal occlusion was suggested.



A. *B.*

Fig. 4.—*A*, Translumbar aortogram performed under local anesthesia giving false impression of terminal arterial occlusion. *B*, Retrograde femoral aortogram performed under spinal anesthesia demonstrating segmental occlusion of left femoral artery and bilateral collateral filling.

Extravasation of Radiopaque Medium.—This is not an uncommon occurrence following the percutaneous method. It is usually associated with sharp and sudden pain at the injection site which often disappears after several

hours. In one case of retroperitoneal extravasation of contrast medium following translumbar aortography, back pain persisted for fully two weeks.

Sensitivity Reaction to Dye.—Known sensitivity to the contrast medium is perhaps the only absolute contraindication to arteriography. We have observed only transient nausea, weakness, or mild urticaria. None of these have been serious, and most could not be classified as true sensitivity responses. Careful questioning concerning iodine sensitivity followed by intravenous injection of 1 c.c. of dilute dye 10 minutes or more prior to the arterial injection are probably important safety measures. In addition to the narcotic, administration of a barbiturate as a premedication is believed to minimize the hazard of sensitivity. Adrenalin should be available for immediate injecton. In some quarters antihistaminics are recommended as part of the premedication.[10]

A statistical survey[2] of intravenous urography revealed one fatality per 153,000 intravenous Diodrast injections due to anaphylaxis. It has been suggested,[11, 17] that the intra-arterial injection is less likely to lead to such reactions than is the intravenous injection.

Embolism Secondary to a Dislodged Atheromatous Plaque.—We have not seen this complication, nor does there seem to be a well authenticated report of such in the literature. However, it is a possibility which is frequently mentioned as a theoretical danger, especially following the percutaneous method. For this reason it is deemed unwise to perform translumbar aortography in patients who exhibit marked calcification in the abdominal aorta in its entire extent.

Dissection Within the Vessel Wall.—The likelihood of this occurrence is clear although the literature does not reveal serious effects of it. Its theoretical danger in the presence of a partially clotted aneurysm, however, is obvious.

Paraplegia or Hemiplegia.—This complication involving one or both lower extremities, and at times associated with bowel and bladder incontinence, is certainly one of the most unfortunate and dreaded sequelae of aortography. Several instances of transient and occasionally permanent paralysis have occurred in some institutions.[3, 9] The mechanism of this complication may conceivably be the direct injection of dye into the spinal cord. It may be due to faulty insertion of the translumbar needle into the intrathecal space by way of the intervertebral cartilage which can distort the needle and alter its direction. The return of cerebrospinal fluid during this phase of the procedure has been reported.[10]

However, in most instances, it is believed the myelitis develops as a result of dye infusion of the major spinal arteries (arteria radicularis magna) supplying the spinal cord above the level of the cona medullaris. This hazard is considered more likely to occur when the contrast medium enters the spinal arteries in rather concentrated form undiluted by blood. There are usually one or two of these greater radicular arteries supplying the lumbar portion of the spinal cord[9] after rising from the dorsal aspect of the aorta between the eighth thoracic and fourth lumbar segments.[16] It would seem that avoiding especially the higher of these vessels, either from the point of view of needle

abrasion or injection of relatively undiluted dye, is important in preventing damage to the spinal cord. In theory, then, this complication would be minimized by performing abdominal aortography: (1) always with the patient in the prone position, (2) by inserting the needle below the level of the cona medullaris, i.e., below L_1 when the translumbar route is used, (3) by injecting no more contrast medium than is necessary, (4) by avoiding excess pressure during injection, and (5) by avoiding multiple injections in rapid succession, preferably waiting a moderate length of time (15 to 20 min.) between injections if a second one is necessary.

Oliguria.—This complication associated with renal shutdown has been frequently described.[2, 14] It has been found to follow the injection of dye into the aorta at or immediately above the level of the renal arteries, especially when there is obliterative disease of the terminal aorta. Unilateral renal shutdown has been described following the direct injection of one renal artery with dye during attempted aortography. Here again it appears that relatively undiluted dye can cause specific tissue damage. To avoid this complication the dye should be injected below the level of the renal arteries (i.e., caudad to the vertebral body of L_1). If it is necessary to visualize the renal arteries, an injection site well above their origin should be chosen to allow dilution of dye. It has also been suggested[10, 18] that arteriography not be performed in the presence of poor renal function, a precaution probably analogous to that observed when considering intravenous pyelography.

Perforation of Abdominal Viscus.—This occurrence is mentioned in the literature,[14, 17] but apparently has not been of serious consequence in experienced hands.

COMMENT

It would be unfortunate indeed if a diagnostic procedure as informative as angiography were not employed when indicated. It is the more unfortunate, however, if the procedure is responsible for catastrophic complications. We have the firm conviction that more careful consideration of indications and contraindications, more meticulous attention to certain technical details, and full realization of the responsibilities involved will lessen the hazards of angiography and at the same time endow it with even more significance than it possesses today.

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DISCUSSION

DR. EDWARD A. EDWARDS (Boston, Mass.).—The use of two maneuvers will often make arteriography unnecessary. The first is oscillometry, which if performed at various levels of the limb may clearly indicate the level of obstruction. The second is auscultation—the finding of a systolic murmur indicating partial but severe obstruction. Thus, if there is lack of pulsation below the femoral level, the presence of a murmur over the iliac artery or aorta attests to additional obstruction at a higher level. The pathology in such a patient is at once shown to be widespread, even without arteriographic examination.

I am much opposed to the proposition that aortography is to be preferred to bilateral femoral arteriography; the hazards of aortography are so much greater.

Finally, a few items relating to the technic of aortography are:

1. We are all perhaps too anxious to produce a superb picture in the aortogram. Adequate visualization may often be obtained through the use of 35 per cent rather than 70 per cent contrast medium. This should make the injection safer, particularly when it must be made at juxtarenal levels.

2. In obstructive disease the needle can usually be inserted below the origin of the renal arteries. This should substantially reduce the incidence of complications, except for the rare effect on the large bowel.

3. A slow, rather than fast, injection of the medium is superior in the delineation of the iliac and femoral arteries when a block exists in the great vessels. Making the x-ray exposure after an injection prolonged over about 8 seconds will allow the material to be carried across the collaterals to the vessels below.

DR. EUGENE OSIUS (Detroit, Mich.).—At the risk of repetition, before some of this group last year we reported an accident. One of our residents inadvertently injected contrast material into the inferior mesenteric artery. We did an abdominoperineal resection as an emergency measure that night. The patient survived.

Another resident, to show what can happen even under supervision and with care, made one attempt and with a quick locating film in which we simply inject a small amount to see if the point of the needle is in one of the smaller or large vessels found it was in the superior mesenteric. Fortunately he had not injected very much contrast material. He tried again and inserted the needle in the inferior mesenteric. He sent the patient back to bed and the following day was successful in obtaining a good aortogram.

DR. JERE LORD (New York, N. Y.).—One more possibility exists. One does everything the best way one knows how, and still the patient may get into trouble.

The patient had a normal arterial circulation to his right foot. He had obstruction of the left external iliac artery. The left femoral pulse was absent. Translumbar aortogram was performed with the needle opposite the lower border of the second lumbar vertebra and 5 c.c. of 70 per cent Urokon was injected. A faint outline of the terminal aorta and of the proximal portion of each common iliac artery was visualized. Also, there was a faint outline of the inferior mesenteric artery. Thirty-five c.c. of 70 per cent Urokon was now injected and the patient experienced severe left lower quadrant abdominal pain in spite of the fact that he had a spinal anesthesia sufficiently high to render the skin of the lumbar region insensitive. The slide showed the result of the second injection. The Urokon outlined the terminal aorta but did not enter either common iliac artery, and the majority of it entered the inferior mesenteric artery and outlined the entire vasculature of the sigmoid and rectum. The patient was placed on a clear liquid diet and neomycin and was observed carefully. During the next 24 hours he had 6 bloody bowel movements and the sigmoid became tender and could be felt as an indurated structure. The next day the pain had completely cleared and the sigmoid was less apparent and he was explored. At operation the sigmoid and rectosigmoid were found to be edematous but in all other respects appeared normal. The entire external iliac artery and superficial femoral artery were found to be thrombosed and therefore no by-pass was attempted. The patient made an uneventful recovery.

DR. HAROLD LAUFMAN (closing).—There are many points that could be mentioned regarding the application of techniques to avoid complications, but there is not time to do it here.

I should like to mention one thing that I think would help considerably, and that is to stimulate the pharmaceutical profession into producing new contrast media for us. I do not think we have the ideal contrast medium today.

I would like to thank all the discussants, Drs. Edwards, Osius, and Lord, and I compliment Dr. Trippel on a very good presentation.

THE VALUE OF LUMBAR SYMPATHECTOMY IN INTERMITTENT CLAUDICATION

JOSEPHUS C. LUKE, M.D., F.A.C.S., F.R.C.S., MONTREAL, CANADA

(From the Surgical Service, Royal Victoria Hospital)

THE symptom of intermittent claudication is one of the most exact in medicine pointing to the pathologic process involved and the level of this involvement. Pain in the foot, calf, thigh, buttock or low back which comes on when walking and is relieved by a short rest is typical of true claudication due to obliterative arterial disease. This symptom can be checked for veracity by the fact that the pain comes on more rapidly on fast walking and is delayed when the gait is slowed. Walking uphill accelerates the onset of pain and downhill increases the claudication distance. Pain felt in the foot usually means main vessel occlusion below the knee; if in the calf the occlusion is usually in the popliteal or femoral arteries or higher; in the thigh the iliacs are involved; and in the low back and buttocks lower aortic occlusion is usually the cause. This diagnostic impression can be confirmed by the clinical examination with determination of the existing palpable pulses and the oscillometric readings. Aortic or femoral arteriograms will give a visual presentation of the exact findings.

Before active therapy is discussed, the fact should be stressed that intermittent claudication (usually calf) can resolve spontaneously. The author has at least ten cases seen initially with calf claudication who showed no palpable pulses below the common femoral and low calf oscillometric readings. In six to eight months the claudication had disappeared, at least one pedal pulse had reappeared (albeit weaker than the opposite), and the oscillometric readings had improved. Most of these recoveries had been spontaneous with no treatment. If such a case had been under any special form of therapy including vasodilator drugs or lumbar sympathectomy, an erroneous opinion of the efficacy of this treatment would have been formed. The answer to this type of case is that the individual developed a very short segmental occlusion in the lower part of the femoral artery which has become by-passed by an excellent collateral circulation.

Every surgeon who has performed many lumbar sympathectomies for occlusive arterial disease of the leg has been agreeably surprised by a few patients who have improved their claudication distance markedly immediately after operation. On the other hand, the majority have shown disappointing results with little or no improvement in their walking ability. It is for this reason that it is the general consensus[1-3] that lumbar sympathectomy in the

Read at the tenth annual meeting of the Society for Vascular Surgery, Chicago, Ill., June 10, 1956.

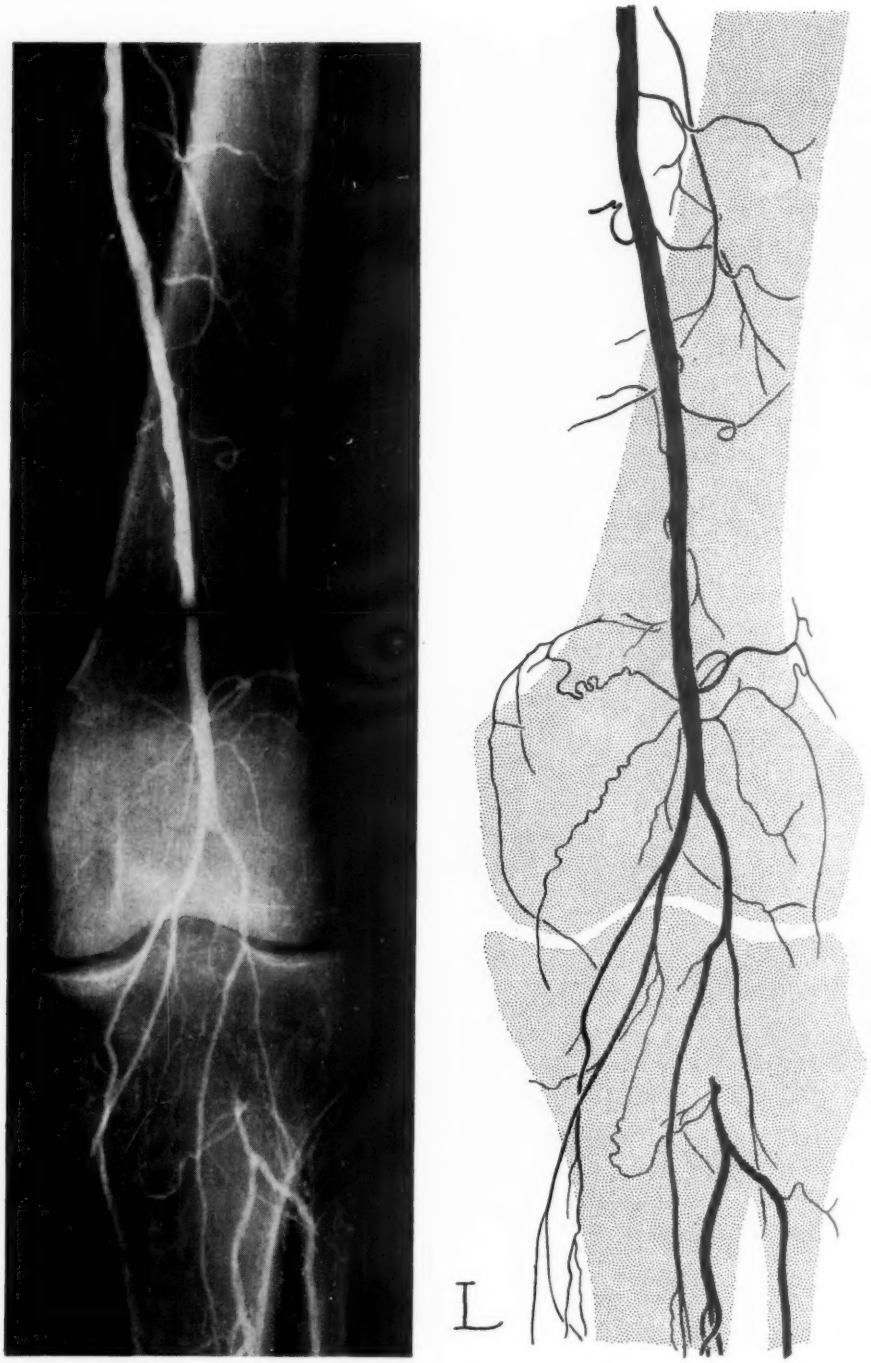

Fig. 1.—Femoral arteriogram showing popliteal artery obstruction. This patient had preoperative calf claudication after two blocks but postoperatively can walk an unlimited distance without pain.

patient complaining only of pain on walking is not a worthwhile procedure. It is in an attempt to reconcile these varying results that this investigation is presented.

In the past three years, all of our patients showing clinical evidence of occlusive arterial disease of the legs whose general condition was satisfactory have had femoral or aortic arteriography as part of their investigation. Only

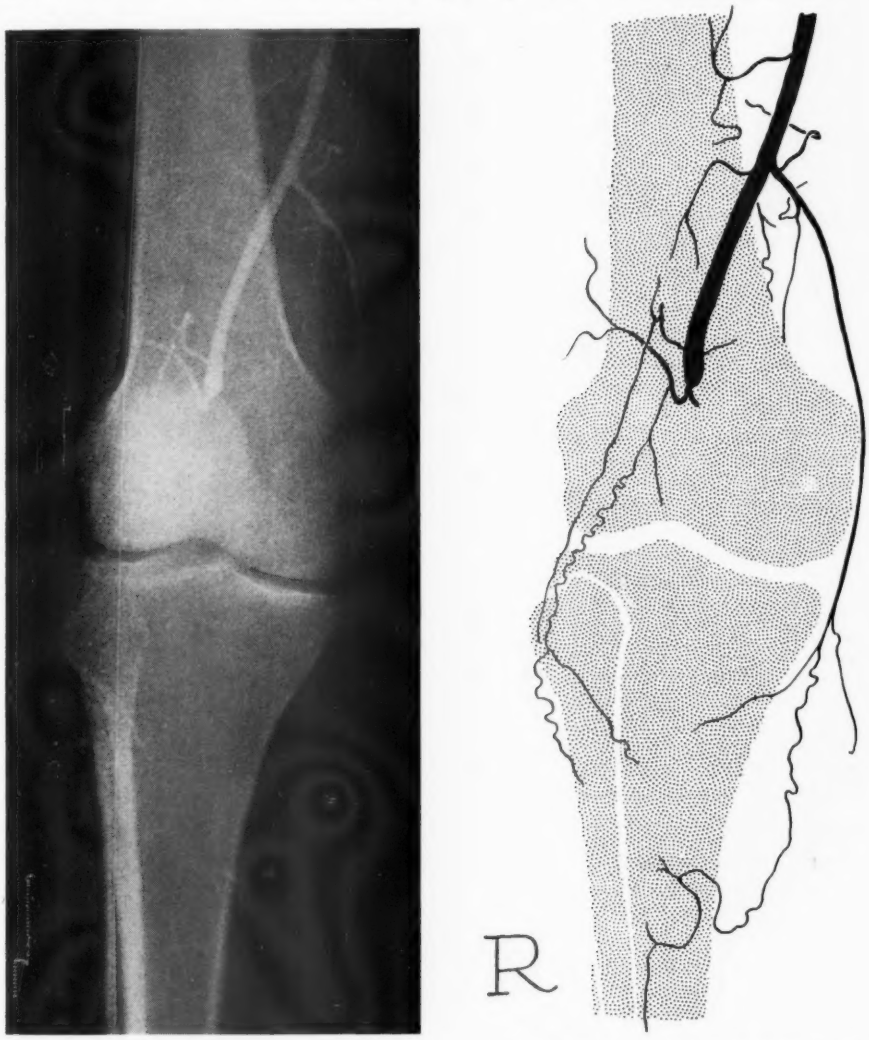

Fig. 2.—Femoral arteriogram revealing popliteal occlusion with limited distal collaterals. Preoperative walking distance, one block: postoperative, four blocks before onset of calf claudication.

one misadventure has occurred in over 300 such examinations. This was a femoral thrombosis which necessitated a supracondylar amputation. Arteriography is done to determine evidence of segmental occlusion which would be benefited by arterial restoration either by thromboendarterectomy or arterial grafting. Seventeen cases complaining of calf claudication revealed arteriograms which showed good filling of the femoral artery down to the popliteal with abrupt termination and no evidence of filling of the main vessels below this level (Figs. 1 and 2). Collateral circulation only extended

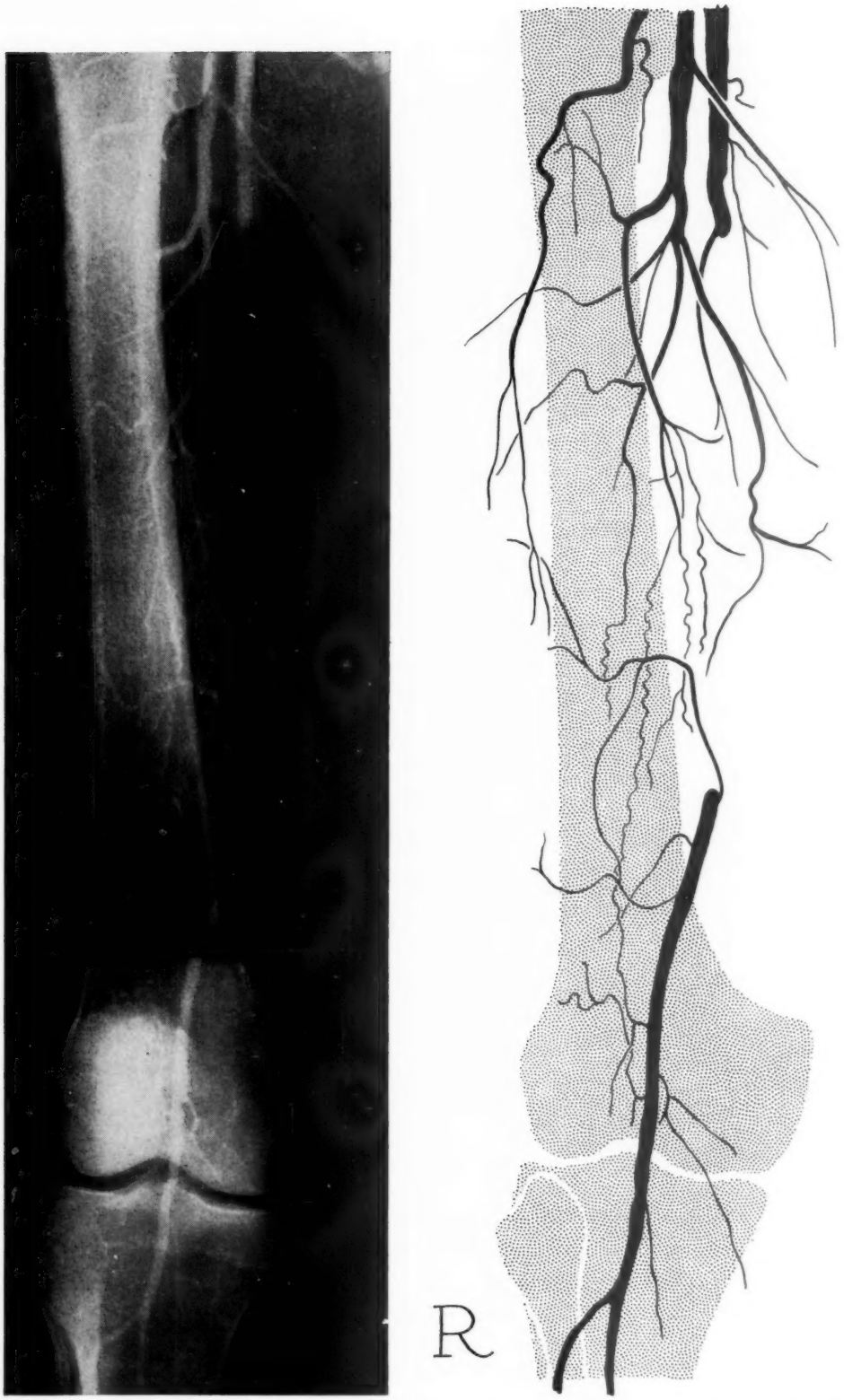

Fig. 3.—Arteriogram showing segmental femoral artery occlusion. This patient was treated by lumbar sympathectomy four years ago before the use of grafts became appreciated. The foot warmed but the claudication distance was little if any improved.

distally. These patients were obviously not suitable for arterial grafts and all were treated by lumbar sympathectomy with removal of the first, second, and third lumbar ganglia. The results of these seventeen cases form the basis of this report.

The marked and, at times, spectacular improvement in walking distance achieved immediately after operation compared to the poor results when the occlusion was higher in the femoral or iliac vessels demanded an explanation. The difference in results according to the different levels of occlusion at once suggested the degree of active collateral vessels as the answer. The more distal toward the popliteal bifurcation the blockage is the more active collaterals with a good head of pressure there are in play. The geniculate branches of the popliteal anastomose with one another and by-pass the popliteal artery connecting with the anterior and posterior tibial arteries or their branches. These geniculate branches are the only detours about an occluded popliteal to supply the muscle mass of the calf. The collateral circulation about a mid or high femoral block, be it a total or segmental occlusion, delivers blood to the popliteal region at low pressure much of which is lost to the knee joint. Improvement of these collaterals by sympathectomy is still insufficient to materially improve the circulatory needs of the calf muscles (Fig. 3).

ANALYSIS OF CASES

In this group of 17 patients with popliteal artery occlusion, all were men; the youngest was 42 years old and the oldest was 67. Two were mild diabetics. The preoperative investigations included blood sugars and cholesterol determinations, electrocardiogram, and femoral arteriogram of the involved extremity. The oscillometric readings at the level of the upper calf were in every instance below 1.0. Lumbar sympathectomy was performed by an extraperitoneal oblique muscle splitting flank incision taking great care that the first, second, and third ganglia were removed. If these ganglia were difficult to identify, the chain length removed extended from well into the crus of the diaphragm to just below the pelvic brim. The postoperative course was uneventful in all cases, and as was expected, the foot warmed markedly. They were discharged from hospital on the seventh or eighth postoperative day. All were warned before leaving that they might expect an aching soreness down the lateral thigh coming on about the tenth postoperative day. All cases experienced this discomfort but only in about half was it of sufficient degree to need sedatives. The more anxious and apprehensive types seemed to have this complication the worst. This postsympathectomy neuralgia is the only bugbear to an otherwise simple operation and appears to be more frequent when the first lumbar ganglion is removed.

Prior to operation the patients walked our hospital corridor which corresponds in length to the city block outside my office. The pace was 120 steps a minute. On follow-up examination between two and three weeks after operation, they were put through their paces on the city block mentioned above. Preoperatively the claudication distance varied from one to four blocks. Postoperatively those with an initial one block distance could do at

least three to four blocks and at that point, although claudication occurred, it was not as severe as formerly. Those with initially better claudication distances achieved better results up to eight or ten blocks, and two cases with an initial four block distance could walk indefinitely without symptoms. The fact that this improvement occurred so soon after operation indicates that the result is surgical and not to the gradual improvement that occurs after most arterial occlusions. This fact would add further clinical proof that sympathectomy also dilates arteries to muscle and not only those to the skin.

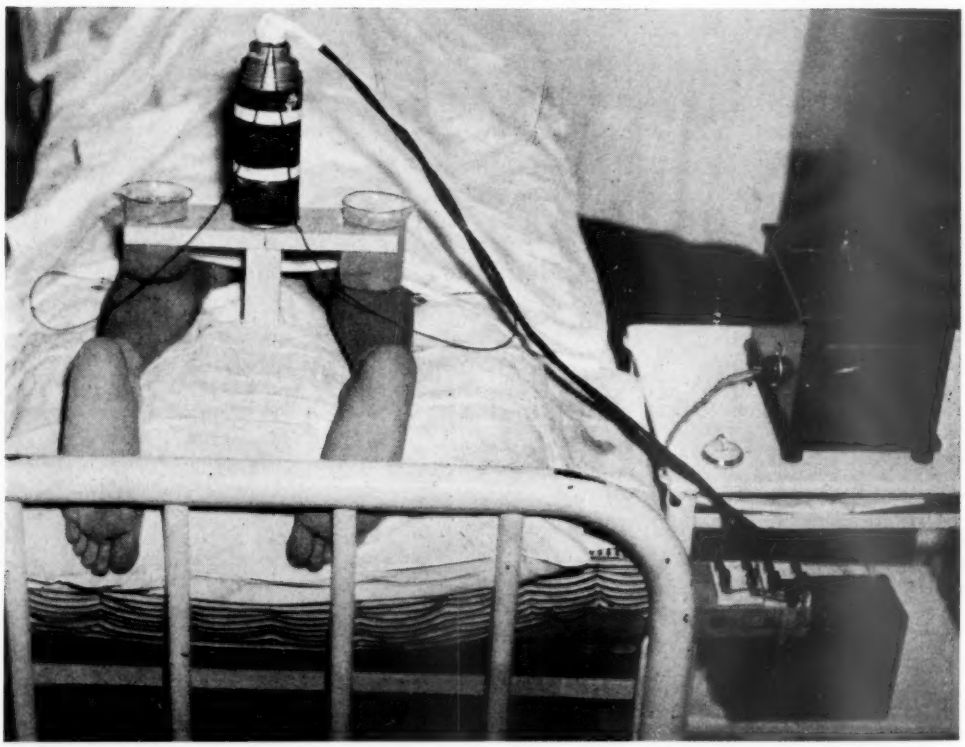

Fig. 4.—Photograph of technique designed to indicate changes in muscle temperature pre- and postsympathectomy. Thermocouples were placed in the gastrocnemius muscle and the rate of cooling from ice water in the overlying beakers was determined in the pre- and postoperative case.

An attempt to prove the increased muscle blood flow in these cases was carried out by Dr. K. A. Evelyn and the author by placing a special thermocouple into the gastrocnemius muscle (Fig. 4) with determination of variation in temperatures before and after sympathectomy. No constant difference in recordings was observed. A further refinement was also employed to see if there was any difference in rate of local cooling between the pre- and postoperative case. A beaker of ice water was placed on the skin over the gastrocnemius one inch superficial to the muscle thermocouple, and the curve rate of cooling was plotted. It was hoped that the rate of cooling would be slower in the postoperative period. However, the cooling graph was variable and did not show a consistently definite trend, and consequently we have no experimental proof of the clinical improvement.

As a result of our findings in these 17 cases we believe that lumbar sympathectomy is a worthwhile procedure in alleviating and sometimes relieving the complaint of intermittent claudication. This is so if the occlusion is in the popliteal artery and the patient has a good femoral artery down to this level. Occlusions above the adductor canal do not give equivalent results and the operation is not justified in such people for intermittent claudication alone. Fortunately these higher obstructions are usually segmental in character and therefore suitable for the procedure of end-to-side arterial by-pass graft.

CONCLUSIONS

1. The different aspects of intermittent claudication due to occlusive arterial disease of the legs are discussed. Seventeen cases are presented where arteriography revealed the occlusion to be present in the popliteal artery. The only complaint was calf claudication in all cases.

2. This group was treated by lumbar sympathectomy with improvement in walking distance threefold or more. In consequence we feel that we can promise individuals with popliteal artery occlusion such an improvement if the first, second, and third lumbar ganglia are removed.

3. This is in contrast to little or no improvement in walking distance when sympathectomy is performed for claudicants whose level of occlusion is higher in the femoral artery. The collateral circulation about the knee is the probable factor in the difference in results between the popliteal and femoral levels.

4. Experimental confirmation of the improvement in muscle blood flow demonstrated clinically in the popliteal group was not definite.

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2. Wright, I.: Conservative Treatment of Occlusive Arterial Disease, Arch. Surg. **40:** 163-189, 1940.
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DISCUSSION

DR. ROBERT LINTON (Brookline, Mass.).—I have enjoyed Dr. Luke's presentation very much, but I disagree with him in regard to the improvement in intermittent claudication following lumbar sympathectomy. In my opinion this operative procedure has its greatest use in augmenting the blood supply to the integument of the foot and toes; therefore I consider it primarily a limb-saving procedure, and so do not tell my patients that their walking ability is going to be improved following a lumbar sympathectomy.

We have been interested in this method of treatment for obliterative arterial disease of the lower extremity for a number of years at the Massachusetts General Hospital. During the past 6 years, we have been running a long-term study to determine the effect of lumbar sympathectomy in patients treated by this procedure and comparing the results with those in a comparable control group without sympathectomy. At the present time, I can give you the results in 19 patients in the control group and 19 in the sympathectomy group which have been studied carefully for over 3 years in regard to their intermittent claudication. It is of interest that 2 of each group were free of claudication, 13 of each were improved, and 4 of each had become worse. These figures speak for themselves and I can assure you the studies on them were carried out as scientifically as is possible in estimating intermittent claudication.

(Society for Vascular Surgery articles will be concluded in the February, 1957, JOURNAL.*)*

Book Reviews

CONDUCTED BY J. GARROTT ALLEN, M.D.

Hypothermic Anesthesia. By Robert W. Virtue. Monograph in American Lectures in Anesthesiology, Springfield, Ill., 1955, Charles C Thomas. Pp. 62.

". . . . This essay should not be considered a summary on hypothermic anesthesia, but should be taken as an interim report on its status." The reviewer cannot but agree with Virtue's foregoing statement, albeit the 84 references are an excellent guide for anyone interested in this subject.

Virtue has compiled an easily readable, complete, yet not too dogmatic brief on the present status of hypothermia. Such an attitude is almost obligatory when one considers changing concepts in the field. The number of problems posed exceeds the answers given, and the author repeatedly stresses that techniques and observations currently are in a state of flux. Further, the author has chosen to stress physiologic changes associated with hypothermia rather than techniques of attaining the state itself.

While the bulk of the monograph is in essence a survey of literature, one chapter is devoted to the author's own methods and experiences, and is particularly instructive when one examines the mortality possibly related to the technique itself.

The book is well bound in black lexide.

—E. A. Schultz

Campbell's Operative Orthopaedics. By J. S. Speed and R. A. Knight. Ed. 3 (Vol. I and II. St. Louis, 1956. The C. V. Mosby Company. $40.00.

This new 2-volume third edition incorporates an extensive revision from both the original classic by Campbell in 1939 and later the first 2-volume edition in 1949.

In reviewing these 2 new volumes one cannot help but realize the magnitude of this new edition. A new chapter on surgical physiology has been added. Included here is the latest work on ketosteroids and fluid balance. The problem of fluid and mineral balance is well detailed yet simplified to such a degree that the uninitiated could orient himself. Also, a concise but informative discussion is given on cardiac arrest and the treatment of this condition. This chapter indeed adds a great deal to the book.

The chapter on surgical approaches leaves little to be desired. Most of the classical illustrations and approaches that have been seen and described before are presented again. The clarity and exactness of most of the illustrations make this chapter truly outstanding; in fact, the quality and profuseness of the illustrations in the entire 2 volumes are to be commended.

Most of the other chapters have been extensively revised deleting many of the older procedures and concepts, concomitantly adding new diagnostic measures and present-day operative techniques. In perusing the entire 2 volumes one cannot help but feel what a tremendous love and inspiration the authors must have had for this momentous task. Besides having a writing style throughout the book that is easily read it is pleasing to note that the organization and presentation of the material are excellent.

In a book of this magnitude one cannot help but find some small discrepancies and debatable issues. However, all in all, there are no glaring weaknesses with which one could take a major issue. It does seem regrettable that a chapter on plastic procedures useful to the orthopedic surgeon has been omitted. The authors acknowledge this omitttnce because

of the great detail that would be necessary for such a review. It is understandable that the plastic field has come of age to the extent that such a chapter might become a bit cumbersome, but a discussion of the plastic procedures useful to the orthopedic surgeon in reconstructive surgery would seem to be of appropriate value and a fitting addition to this fine book. The omission of contributions from the foreign literature leaves a void in some procedures which might add to the armamentarium of the orthopedist. However, it is gratifying to note that this weakness has been recognized and undoubtedly will be corrected at a later date.

One might take issue with the anterior approach to the knee joint as described by Coonse and Adams in which the quadriceps tendon is divided. The authors have noted that this exposure is best for arthrodesis of the knee where re-establishment of quadriceps motion is not necessary. It seems that this point should be re-emphasized and the approach should be strongly advised against in which knee joint function is desired as there are more desirable approaches which will allow adequate exposure and still permit excellent return of motion and strength.

Suggestion has been made that primary arthroplasty should be carried out in some cases of fracture dislocation of the hip. It is conceivable that an occasional case in which the continuity of the femoral head has been completely destroyed would be a logical candidate for such a procedure. However, if the majority of the head is intact, conservative management for the first 1 or 2 years seems to be indicated. Several pages have been allowed to the technique of taking x-rays of the hip in the operating room, and yet omitted is the technique in common use today where the plate is placed above the hip joint with the x-ray tube between the legs. This method has been in the reviewer's experience a very satisfactory and less cumbersome procedure than the technique described where the tube is placed superior to the hip joint and a curved cassette is used between the legs.

One could continue to discuss personal preferences and debatable issues, but as a rule the authors have presented alternative techniques when there is a choice in question. Yet, the authors have been straightforward in describing an accepted technique in order that the novice will have one good procedure to follow if his judgment has not matured to a point where he may select one of several methods.

The bibliography is extensive and includes most of the leading articles on the subject discussed. The authors are to be complimented on this momentous task of making the extensive revisions that have been placed in this third edition. It is hoped that the general acceptance which is certain to be given this book will in some way reward the authors for the Herculean effort that has been put forth. The reviewer feels that this 2-volume edition should be in the hands of every orthopedic surgeon in practice today and available to any man who has an occasion to delve into the orthopedic field.

—Harry B. Hall

Pre- and Postoperative Care in the Pediatric Surgical Patient. By William B. Kiesewetter. Chicago, Year Book Publishers, Inc. $7.00.

Kiesewetter has assembled in this 334-page book the contributions of 15 of his colleagues at the University of Pittsburgh School of Medicine. Despite the fact that in the last 2 decades there are nearly 40 monographs or texts on this subject, most of which deal with fluids and electrolytes, this manual should fill a need not yet met. Its purpose is to outline in some detail various aspects of pre- and postoperative care in the field of pediatric surgery, being addressed to the "occasional pediatric surgeon." The principles set forth are those employed at the Children's Hospital of Pittsburgh.

The first third of this manual is devoted to subjects such as fluid and electrolyte balance, anesthesia, and postoperative considerations as they pertain to the infant or child. This contains very useful information, such as dosages of drugs, fluid requirements, growth, weight, and surface relationship, which should serve to orient the surgeon whose practice only occasionally involves the child or infant. Again at the end of the book these subjects are summarized in a practical fashion, with tables and formulas spelling out specific practices.

More than half of the pages deal with specific pre- and postoperative considerations in outline form as encountered in the various fields of surgery: abdominal, thoracic, orthopedic, neurologic, plastic, urologic, otorhinolaryngologic, and ophthalmologic. In each, the more commonly encountered conditions are outlined as to "required studies, elective studies, differential considerations, preoperative treatment and orders, and postoperative management." Such a scheme, of course, invites some errors of omission and of interpretation, such as leaving out blood and/or plasma loss as a cause of shock, although the use of these fluids in its treatment is well covered on the next page. On the whole, this volume skirts such hazards remarkably well. Bibliography is brief which may detract to some extent from its value; the classical work of the pediatrician, James Gamble, is not included in the chapter on fluid and electrolyte balances.

Nonetheless, this manual does serve as a ready source of material which should be useful to the medical student, house staff, general practitioner, and the occasional pediatric surgeon.

—J. Garrott Allen

Cancer of the Esophagus and the Stomach. By Owen H. Wangensteen. Ed. 2. New York, 1956, American Cancer Society, Inc.

This is an excellent and succinct presentation of the cancer problems affecting the esophagus and the stomach, designed to assist in the early recognition of cancer in these organs. The approach employed by the author bears not only upon diagnosis and treatment but also upon frequency, location, predisposing factors, pathology, mortality, and survival rates. Historical references are briefly alluded to, which add materially to the reader's interest.

Of special interest are the improved surgical results for carcinoma of the stomach in terms of 5-year survival in a number of major clinics in this country and abroad and, in particular, a rather extensive review of the Minnesota experience. It is encouraging to note that 57 per cent of patients subjected to gastric resection for carcinoma of the stomach without involvement of the lymph nodes survived at least 5 years. But it is discouraging to realize that only 14 per cent of those with nodal involvement survived similar periods. Moreover, Wangensteen states that essentially 75 per cent of all patients with gastric carcinoma have no metastases at the time of operation and that this percentage of nodal involvement has not been materially reduced during the past few decades.

The "second-look" procedure for gastric carcinoma, Wangensteen points out, has been less useful than when applied to patients whose cancer arises within the colon. Fifty-eight per cent of patients with gastric cancer who had metastatic carcinoma in their lymph nodes at the time of their first operation were found to have residual cancer at the time of the "second-look" procedure. The predominant location of recurrences for gastric cancer was in the gastrohepatic omentum.

This little monograph is full of factual information and should be of great interest to surgeon and internist alike. It may be obtained free of charge from the American Cancer Society, 444 East 68th Street, New York, New York.

—J. Garrott Allen

The Initial Management of Thoracic and Thoraco-abdominal Trauma. By Lawrence M. Shefts. Springfield, Ill., 1956, Charles C Thomas.

This is an excellent book covering the management of chest wounds and combined chest and abdominal wounds. Emphasis upon "initial" indicates the major thesis of the book as that of resuscitation and general management with surgery recognized as adjunctive, to be considered seriously and carefully timed; it also allows deletion of delayed complications and therapy. There is solid clinical information throughout the entire book, clearly based upon experience in living with, nursing, and operating upon the wounded soldier as Dr. Churchill points out in the Foreword. Resuscitation is thoroughly described

in the first chapter with practical details and illustrations down to bandage shape, needle size, and tubing care. Attention to details and constant appraisal of response to the resuscitation are emphasized. The second chapter on Thoracic Wounds presents mortality data, missile data, and the importance of judging the course of missiles in the body. Indications for surgery are handled forthright and completely, noting, for example, how carefully one must judge continued intrathoracic bleeding as an indication for thoracotomy. There is a long historic and physiologic discussion of cardiac injuries, probably because the author in only this instance had need for the experience of others to outline adequately his treatment. Esophageal trauma proves to be a small hazard, but the care of this is described, and some people may disagree with the painful mediastinotomy drainage recommended in right-sided injuries when there has been a necessary thoracotomy. Thoracoabdominal injuries are treated with reference to side (emphasizing the great dangers of left-sided injuries) perforating or penetrating. This is followed by thoracic wall wounds and closed wounds including the important ruptures of the diaphragm and flail chest (''mandatory tracheotomy'').

Important similarities to civilian accidents and atrocities are given. This book should be read by all practitioners and resident staff who handle any amount of accident cases, because this is an area of great need for clear-cut methodology as is so well presented in this book. This is a need second only to that of all service physicians and surgeons. Such books as this should allow a continued train of knowledge of war casualty studies not hidden in Service Archives so that we are not forced to relearn all of the past with each war or ''policing'' effort. Licensed by his title, *Initial Management*, Dr. Shefts dodges the controversial, ever-changing issue of antibiotics by never mentioning them.

—Peter V. Moulder

Calendar of Surgical Meetings

1957

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY. *Annual Meeting,* May 4, 6, and 7, 1957, The Palmer House, Chicago, Ill. Deadline for papers, Dec. 15, 1956. Secretary, H. T. Langston, M.D., 1919 West Taylor St., Chicago 12, Ill.

AMERICAN ASSOCIATION OF GENITO-URINARY SURGEONS. *Annual Meeting,* May 1, 2, and 3, 1957, The Homestead, Hot Springs, Va. Secretary, John A. Taylor, M.D., 2 East 54th Street, New York 22, N. Y.

AMERICAN ASSOCIATION FOR THE SURGERY OF TRAUMA. *Annual Meeting,* Oct. 31, Nov. 1 and 2, 1957, The Homestead, Hot Springs, Va. Deadline for papers, Aug. 1, 1957. Secretary, James K. Stack, 700 N. Michigan Ave., Chicago 11, Ill.

AMERICAN COLLEGE OF SURGEONS. *Sectional Meetings,* 1957: Jan. 16-18, Caribe Hilton, San Juan, Puerto Rico; Feb. 4-7, Hotels Jung and Roosevelt, New Orleans, La.; Feb. 28-March 2, The Olympic Hotel, Seattle, Wash.; March 18-20, The Sheraton-Park Hotel, Washington, D. C.; March 25-27, The Royal York, Toronto, Ontario. *Clinical Congress,* Oct. 14-18, Convention Hall, Atlantic City, N. J. Secretary of the College, Michael L. Mason, 154 E. Erie St., Chicago 11, Ill.

AMERICAN COLLEGE OF SURGEONS. Southern California Chapter. *Annual Meeting,* Jan. 18, 19, and 20, 1957, Biltmore Hotel, Santa Barbara, Calif. Secretary-Treasurer, Max R. Gaspar, M.D., 211 Cherry Avenue, Long Beach 2, Calif.

AMERICAN GYNECOLOGICAL SOCIETY. *Annual Meeting,* May 27, 28, and 29, 1957, The Homestead, Hot Springs, Va. Secretary, Andrew A. Marchetti, M.D., Georgetown University Hospital, Washington 7, D. C.

AMERICAN MEDICAL ASSOCIATION. *Annual Meeting,* June 3-7, 1957, Coliseum, New York, N. Y. Deadline for papers, Dec. 15, 1956. *Clinical Meeting,* Dec. 3-6, 1957, Auditorium, Philadelphia, Pa. Deadline for papers, July 15, 1957. Secretary, American Medical Association, George F. Lull, M.D.; Secretary, Council on Scientific Assembly, Thomas G. Hull, M.D.; address of both, 535 North Dearborn Street, Chicago 10, Ill.

AMERICAN SURGICAL ASSOCIATION. *Annual Meeting,* May 8, 9, and 10, 1957, The Palmer House, Chicago, Ill. Secretary, R. K. Gilchrist, M.D., 59 East Madison Street, Chicago 3, Ill.

AMERICAN UROLOGICAL ASSOCIATION. *Annual Meeting,* May 6-9, 1957, William Penn Hotel, Pittsburgh, Pa. Deadline for papers, Jan. 1, 1957. Secretary, Samuel L. Raines, M.D., 188 S. Bellevue Blvd., Memphis, Tenn.

THE CENTRAL SURGICAL ASSOCIATION. *Annual Meeting,* Feb. 21-23, 1957, The Drake Hotel, Chicago, Ill. Deadline for papers, Oct. 15, 1956. Secretary, Charles D. Branch, M.D., 102 North St., Peoria, Ill.

THE HARVEY CUSHING SOCIETY. *Annual Meeting,* April 25-27, 1957, Hotel Statler, Detroit, Mich. Secretary, David L. Reeves, M.D., 316 W. Junipero St., Santa Barbara, Calif.

NEW ENGLAND SURGICAL SOCIETY. *Annual Meeting,* Oct. 4 and 5, 1957, Mount Washington Hotel, Burton Woods, N. H. Deadline for papers, June 1, 1957. Secretary, Richard Warren, M.D., Peter Bent Brigham Hospital, 721 Huntington Avenue, Boston 15, Mass.

THE PACIFIC COAST SURGICAL ASSOCIATION. *Annual Meeting,* Feb. 4-6, 1957, El Mirador Hotel, Palm Springs, Calif. Secretary, Carleton Mathewson, Jr., M.D., Stanford University Hospital, San Francisco 15, Calif.

PAN-PACIFIC SURGICAL ASSOCIATION. *Annual Meeting,* Nov. 14-22, 1957, Honolulu, Hawaii. Director General, F. J. Pinkerton, M.D., Room 230, Young Building, Honolulu, Hawaii.

THE SOCIETY OF UNIVERSITY SURGEONS. *Annual Meeting,* Feb. 7-9, 1957. the Deshler-Hilton Hotel, Columbus, Ohio. Abstracts to be received by Dr. Ben Eiseman, Chairman, Committee on Publications, 4200 E. Ninth Ave., Denver 20, Colo., by Nov. 15, 1956. Secretary, James D. Hardy, M.D., Hospital of the University of Mississippi, Jackson, Miss.

THE SOCIETY FOR VASCULAR SURGERY. *Annual Meeting,* June 2, 1957, Hotel Biltmore, New York, N. Y. Secretary, Henry Swan, M.D., 4200 E. 9th Avenue, Denver, Colo.

Notice

Because of the large number of original communications awaiting publication in SURGERY, case reports are no longer being accepted.

—THE EDITORS.

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